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THE
LARYNGOSCOPE.

VOL. LXII MARCH, 1952. No. 3

REVISED ANATOMY OF THE RECURRENT LARYNGEAL NERVES. SURGICAL IMPORTANCE BASED ON THE DISSECTION OF 100 CADAVERS.

A PRELIMINARY REPORT.*†

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and
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In 1948, Dr. Brien T. King, of Seattle, Wash., published the results of a series of special dissections and experiments he had been conducting, the results of which have seemed to raise the shroud of mystery and unexplained phenomena from certain phases of laryngeal behavior, more especially the behavior of the larynx following damage to one or both recurrent nerves. Dr. King's ideas so stimulated us that we undertook our own separate investigation of this subject.

This subject has interested many others before us, and, indeed, the older literature is replete with ancient accounts and theories of the course and distribution of the recurrent nerves. For the most complete recapitulation of the historical background to the observations made relating to the larynx, the student of history is referred to a fascinating article which appeared in the *New York Medical Journal* under date of July 9, 1887, by Dr. Franklin H. Hooper.[1]

*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, Calif., Jan. 19, 1952.

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Dr. Hooper's introductory paragraph is worthy of being quoted directly:

"Notwithstanding the centuries that have elapsed since the reputed discovery of the recurrent laryngeal nerves by Galen, it is doubtful if there be another pair of nerves in the body which have given rise to so many diverse and fanciful theories to account for the anatomical peculiarity of their course or to elucidate their physiological functions. At the present day our knowledge of the anatomy of these nerves is, for the most part, complete and exact. The reason why they are not given off by the pneumogastrics at a point opposite the larynx so as to reach that organ by the shortest route, instead of branching from the pneumogastrics low down in the neck and running a complicated and recurrent course to the larynx and recurrent course to the larynx, is now clearly understood. Yet Magendie, in his lectures on the nervous system, delivered at the Collége de France at so recent a date as 1836, asserted that this was a problem which anatomists were unable to solve. But several years prior to the date of Magendie's lectures two articles had appeared in a Scotch journal which, had they come under his notice, would have given him a clue to the explanation of the matter."

The articles to which Hooper referred were published in the *Edinburgh Medical and Surgical Journal* — one by Stedman, in 1823, and one by Hart, three years later, both of which dealt with the origin and course of the recurrent nerve in cases of anomalous right subclavian arteries.

The earliest studies on the recurrent laryngeal nerves appear to be attributed to Galen, Vesalius, and Rufus, the Ephesian. Their attention was directed primarily to the relation of these nerves to the voice. (They found that the voice was lost if these nerves were cut or included in a ligature.)

The very earliest, and, therefore, undoubtedly the very first allusion that the recurrent nerve might be a divided nerve was made by Meckel (whose diverticula will never permit him

*Magendie was the first to demonstrate experimentally the true action of the cricothyroid muscle.

to be forgotten by students of medicine) in "A Manual of Anatomy" under date of 1832. Here, Meckel says that the recurrent nerve is "sometimes double, and, though rare, when this occurs it is always on the right side."

Meckel further considers it probable "that the recurrent nerve results from the plexiform division of the trunk of the pneumogastric, and that its existence is connected with the primitive shortness of the neck, since the larynx is much nearer its origin in the early periods of life than subsequently."

The last two statements have an especial appeal to the authors, about which we shall say more later.

There seems to be a substantal hiatus in the interest of anatomists and clinicians in the subject of the recurrent laryngeal nerves, for there is a wide gap in the literature on this subject extending from the end of the nineteenth century until shortly after World War I.

In 1921, Dilworth[2] made some very sage observations on the recurrent nerve, but missed by only the narrowest margin the true implication of his discoveries and ideas. He stated that "about an inch before it enters the larynx it divides into two divisions." He elaborated on it further only to say that one branch (the smaller) unites in the larynx with the internal laryngeal nerve, while the other (the larger) is the muscular division of the nerve. He did not specifically state that one is sensory and the other is motor, but this seems to be implied.

In the light of our present knowledge of this subject, the important observation obviously was that he saw the two divisions of the nerve.

In a highly intuitive mood, he suggested that the laryngeal nerves are really a plexus of nerves of a highly modified type, but, nonetheless, conform to the pattern of plexuses of the vagus neve such as the cardiac plexus, celiac plexus, etc. With the latter view, the authors are in agreement, since this concept lends itself to the anatomical explanation of a multiplicity

of branches of the recurrent nerve rather than to the iconoclastic teaching that the recurrent nerve is always a single trunk nerve.

Fowler and Hansen[3] laid considerable stress on the vulnerable point in the neck during the course of operations on the thyroid gland where the recurrent nerve might most likely be damaged. These authors state that "in a large majority of cases the most intimate relation between the gland and the nerve was not at the lower pole as is generally assumed, but rather at the posterolateral surface at the junction of the middle and lower third, right at or just above the point where the chief branches of the inferior thyroid artery enter the gland." With this statement we are in full agreement. They further state that the nerve is often in extremely intimate relationship to the gland substance on its posterior surface, an observation which coincides with our own.

We take this opportunity to underscore and emphasize and direct specific attention to the observations of Fowler and Hansen, in 1929, cited above, that the vulnerable point, the point at which the recurrent nerve, completely or partially, may likely be damaged, is at the point where it is in intimate relationship to the inferior thyroid artery itself or one of its branches. In the presence of damage to the artery or one of its branches, immediate brisk hemorrhage results which cannot be controlled by sponging. The application of clamps to control the bleeding will almost surely result in complete or partial damage to the recurrent nerve. A perusal of the accompanying diagrams (see Figs. 1 and 2), which are graphic representations of the branching pattern, not only of the inferior thyroid artery but also its relation to the recurrent nerves, will convince the reader of the many opportunities for damage to the nerve where it lies in close relation to this vessel.

Berlin and Lahey,[4] in 1929, reported on dissections of the recurrent nerves, and Berlin[5] again, in 1935, published a further report on this structure. We are in disagreement with the concept that the recurrent laryngeal nerve ever penetrates the substance of the thyroid gland. We have seen instances

where adenomas have appeared to grow or curve around the main trunk of the recurrent laryngeal nerve, but careful dissection has proven that the nerve did not penetrate the gland substance proper, even though it was obviously in close relationship to the gland. Embryologically, we consider that it is

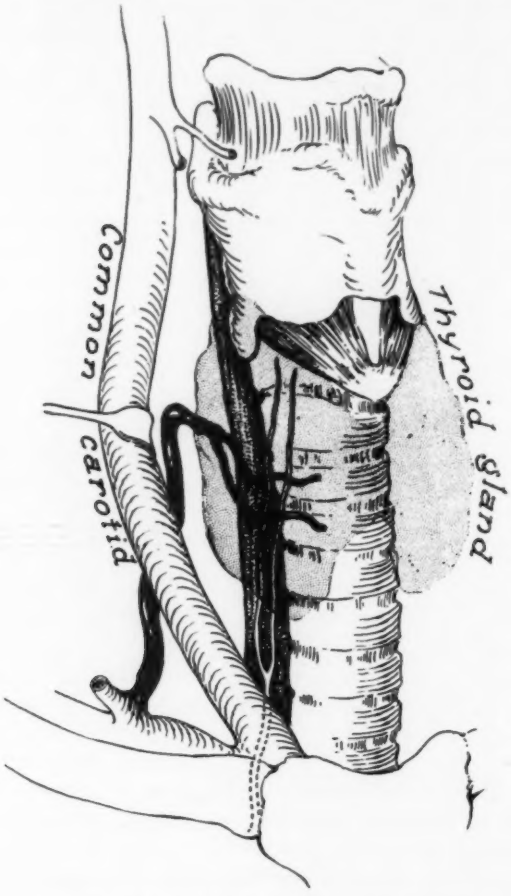

Fig. 1. Divided right recurrent laryngeal nerve.

impossible for the recurrent nerve to traverse the gland parenchyma. We, however, are in agreement with these authors that the inferior cornu of the thyroid cartilage represents a good landmark for the location of this nerve, since the recurrent nerve generally passes just posterior to this articulation.

Fig. 2. Divided, branched left recurrent laryngeal nerve.

We are of the very firm opinion, and, therefore, in agreement with Nordland, that there are no constant differences in

relationship that may be firmly depended upon in the performance of thyroid surgery; that one may find a nerve that has branched two or more times on one side, while finding a single trunk on the other side. Furthermore, these branches or principal trunks may, and often do, intertwine between branches of the inferior thyroid artery and at one point lie anterior to it, while only a centimeter or two away they are lying directly posterior to the vessel.

The meat of the discussion, we believe, is that the recurrent nerve is either well out of the way, lying far posteriorly to the inferior thyroid artery, or is closely associated with it, even almost connected by thin connective tissue strands of neurilemma and adventitia.

In this immediate proximity, it matters little which one lies on top, for in the presence of hemorrhage, the nerve is almost surely to be damaged. This obviously brings up the wise procedure of ligating the main trunk of the inferior thyroid artery far laterally and, therefore, minimizing or eliminating entirely the matter of accidental hemorrhage from this vessel.

Nordland[6] is another observer who depicted the divided or branched recurrent nerve without enlarging on it, and apparently failing to appreciate its significance. In one of his diagrams, a divided recurrent nerve is clearly pictured. Underneath the diagram appears the caption, "the nerves branch around the artery," showing a divided recurrent nerve intertwining between branches of the inferior thyroid artery. One cannot help but wonder how many times this same observation has been made and its significance not recognized or reported. When one considers that such an expert and competent observer as Nordland pictures a recurrent nerve as a divided nerve, and yet describes it as a single trunk nerve, it seems reasonable to assume that other good and noble men have overlooked the possibility for branching of the recurrent nerve simply because we have thought traditionally for generations that the nerve is always a single trunk nerve.

In 1938, Lahey and Hoover[7] also mentioned "bifid nerve" and went on to state that it was unusual, and made no fur-

ther comment. Here again is another example of observers qualified in the highest sense of the word making no more of a point of the presence of a divided recurrent nerve than to state that it was unusual. The authors here feel that it would be extremely easy to miss the presence of a divided nerve in the live patient during the course of operation, for an operator quite naturally, upon discovering a recurrent nerve trunk, would presume this to be the nerve *en toto* and would terminate its dissection rather than lay open all the tissue spaces in the neck in search of a possible second or third branch. It is, therefore, only in the cadaver where the entire area extending from the prevertebral fascia all the way anteriorly to the pretracheal fascia and extending also from a point behind the clavicle and first rib well up onto the thyroid cartilage, that one is able to demonstrate clearly the absence or presence of a divided recurrent nerve.

We have considered the recurrent laryngeal nerve to be a single trunk nerve when it ascends from the thorax, traverses the neck, and enters the larynx without having branched at any point (with the exception of tracheal, esophageal, etc., branches). We have considered the nerve, on the other hand, to be a divided nerve if the point of division was extralaryngeal, regardless of the point at which it divided. For example, we have seen the recurrent nerve divide into two branches immediately (0.5 to 1 cm.) prior to entering the larynx, and we have also seen the recurrent nerve dividing into two or more branches low down in the neck, as low as a point just at the clavicular level. We have seen no nerve trunks divided below the clavicular level.

The classical anatomical descriptions of the origin and course of the recurrent laryngeal nerves bear repetition. The left recurrent laryngeal nerve is derived from the parent vagus nerve on the same side and leaves the vagus nerve at a point just below the arch of the aorta. The nerve courses around the arch of the aorta and assumes a cephalad direction. It tends to approach the trachea and esophagus rather early and, therefore, at the root of the neck the left recurrent nerve generally lies closer to the midline than does the right.

The nerve continues its cephalad course in the tracheoesophageal groove as a single trunk, and after coursing well posteriorly to the thyroid gland, it ascends alongside the cricoid cartilage and enters the larynx in the immediate vicinity of the inferior cornu of the thyroid cartilage.

The right recurrent laryngeal nerve, like its homologue on the left side, is also derived from the main trunk of the right vagus nerve. It leaves the parent nerve at the level of the right subclavian artery and, therefore, hooks around this vessel and begins its ascending course. The right recurrent laryngeal nerve is universally described as occupying a slightly more lateral position with relation to the midline than the nerve on the left side. It, therefore, approaches the tracheoesophageal groove from this somewhat lateral position and ascends to enter the larynx in the same manner as the nerve of the opposite side. This, then, constitutes the description of the origin and course of these nerves as it has been handed down from generation to generation.

We have found that this uniformity of pattern does not exist with so comforting a degree of consistency. In 24 per cent (see Table 1) of our dissections the nerve has been found

TABLE 1.

Bilateral single trunk nerves	35 per cent
Bilateral divided trunk nerves	24 per cent
Mixed single and divided trunk nerves	41 per cent

to be bilaterally divided into two or more branches; furthermore, we have observed no consistency whatsoever in the nerve patterns between right and left sides. To cite a few examples, our experience has not shown that because an individual has a single trunk nerve on one side, of necessity he has a single trunk nerve on the opposite side. Conversely, the presence of a branched nerve on one side does not indicate that he may have a branched nerve on the opposite side. Nor does the presence of a nerve divided into two branches on one side indicate that he may have a nerve divided into two

branches on the opposite side. Indeed, he may have four or five or even six branches on the opposite side. We are not in a position to hypothesize as to the possible number of branches which conceivably could exist, but in the course of our dissections we encountered one specimen in which the recurrent nerve had divided extralaryngeally into six tiny filaments, all of which were seen to enter the larynx between the cricoid cartilage and the thyroid cartilage; therefore, we can only say that we have seen branches of this nerve ranging from one to six.

By the same token, we have observed a completely variable level of division of the main trunks of the recurrent nerve. We have seen this nerve break up into two or more filaments immediately before entering the larynx, which is to say within the range of a centimeter or less; we have also seen the main trunk of the recurrent laryngeal nerve divide into two branches as low down as a point just behind the sternoclavicular junction. We have seen these two branches redivide at only a slightly higher level so that three or four secondary branches have actually traversed the space normally occupied by the thyroid gland. The recurrent nerve, whether as a main single trunk or whether broken up into a variable number of component branches, has entered into many and diverse and completely unpredictable relations with the inferior thyroid artery. Since the recurrent nerve that lies far posteriorly and well out of harm's way is never the nerve that gets injured, we are primarily interested in the neurovascular patterns which are in intimate relationship one with the other.

From a hand to hand acquaintance with the many configurations which are possible in any given patient, it is not hard to appreciate that real hazard exists to the recurrent laryngeal nerves in the performance of thyroid and neck surgery, but that an appreciation of the possible configurations will go a long way to minimizing the likelihood of damage to these structures. The authors believe that the best attitude a surgeon can have is the foreknowledge that the recurrent nerves themselves, and more especially their relationship to the blood

supply of the thyroid gland, is a completely variable one and no reliance may be placed on any classical description which assumes these structures to bear a constant relationship.

We are also of the opinion that single filaments of a nerve with multiple branches are damaged more frequently than is supposed. The reason that these injuries remain undiagnosed is due to the fact that the function of the vocal cords has not been grossly interrupted and, therefore, they assume no position of paralysis which may be observed by a laryngoscopy; however, the complaint of voice fatigue, we feel, is probably indicative of these injuries.

The authors cannot recommend or emphasize too strongly their recommendation that all patients upon whom thyroid or neck surgery is contemplated be subjected to preoperative and postoperative laryngoscopy. While we feel ever so strongly about the advisability and necessity of such an examination, our feelings are doubled in those cases where the patient has been operated upon before for some neck lesion. The medical-legal aspects of such a precautionary examination become clear when one sees malposition or malfunction of one or both vocal cords prior to surgery.

The question might be quite reasonably placed at this time, "How does one avoid damage to an anatomical structure so obscure and so variable?" The authors are of the opinion that the best way to avoid damage to so variable an anatomical structure lies first in the awareness on the part of the surgeon of the multiple anatomical possibilities of the structures involved. It is self-evident that a surgeon cannot look at the neck of an individual and divine whether the recurrent nerve lies in front or in back of the inferior thyroid artery or whether it has one branch or many branches, but if he enters the operating theater with the confidence which emanates from a real appreciation of the anatomy which the area to be operated upon presents, he will in the course of his own dexterity and educated watchfulness be able to carry out his dissections in such a way as either to stay away from danger areas or, realizing that he is in an area which may potentially

result in damage to the recurrent nerves, carry out his dissection in such a meticulous fashion as to avoid accidental bleeding and blind application of clamps and injudicious use of mounted sutures. We realize that there will always be instances where injury to the recurrent nerve cannot be avoided.

There are those who feel that the routine dissection of the recurrent laryngeal nerves at the time of operation is the best method of prevention of damages to these structures. Once again, perusal of the accompanying diagrams will serve to justify the conclusion that it is only in the cadaver that a sufficiently wide dissection may be made, ranging all the way from the clavicle to a point at the top of the ala of the thyroid cartilage. We do not feel that it is practical to make such a wide dissection to isolate completely the recurrent nerves, and yet our dissecting room experience clearly shows that one may be deceived into believing he has dissected out the recurrent nerve when actually he has only one branch of the nerve. A wider dissection would reveal the presence of one or more branches. Additionally, the complete dissection of the recurrent nerves, in order to avoid damage to these structures, is attendant with a certain amount of troublesome bleeding and it is impossible to carry out such a dissection without severance of multiple branches to the trachea and esophagus. It is problematical just how important these small branches might be, but it seems difficult to justify their indiscriminate severance in order to expose completely the entire substance of the recurrent nerve; therefore, though it is repetitive to make the point again, the emphasis cannot be overstressed that a thorough acquaintance with the anatomical possibilities and an appreciation of the danger areas will enhance the success and likelihood of an atraumatic surgical procedure.

CONCLUSIONS.

1. The demonstration of normal division of the recurrent nerve in the thyroid area explains the variations in paralysis produced by surgical or other trauma to this area.

2. This division may occur at any point from the level of the clavicle to the cricothyroid articulation.

3. The nerve pattern is seldom the same on the two sides.

4. The number of extralaryngeal divisions observed ranges from one to six.

5. There is no fixed pattern of relationship between the recurrent nerve and the inferior thyroid artery or their branches.

6. It is evident from our dissections that exposure of a segment of the recurrent nerve, as is possible during a routine thyroidectomy, does not necessarily mean that all of the nerve has been isolated.

7. The results of this anatomical study confirm the contributions made by Dr. Brien T. King.

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OTOLOGIC EVALUATION OF UNILATERAL ACOUSTIC NEUROFIBROMA:

REVIEW OF 150 CASES.*

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Tumors of the cerebellopontine angle present a frequent challenge to the otologist. The VIIIth cranial nerve, extending 17 to 19 mm.[1] from its brain stem connection at the junction of the pons and medulla to the internal auditory meatus, is particularly prone to injury from tumors in this region.

Disturbed function of the VIIIth cranial nerve is the most common single finding in tumors of the cerebellopontine angle. Henschen[2] reported deafness as the first symptom in 60 out of 70 cases of acoustic neurofibroma. Cushing[3] in his excellent monograph on tumors of the nervus acusticus found deafness the initial complaint in 25 out of 30 cases. Cushing stressed the importance of chronology of symptoms in instances of acoustic neurofibroma and stated, "The clinical diagnosis of acoustic neurofibroma can be made with reasonable assurance only when auditory symptoms definitely precede evidence of involvement of other structures in the cerebellopontine angle. Hence when initial symptoms of a growth obviously situated in the angle have another sequence with secondary acoustic symptoms, the diagnosis must remain uncertain until the lesion is exposed. An uninterrupted progress of deafness from irritation to complete or nearly complete deafness helps differentiate acoustic neurofibroma from other angle tumors."

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Meningiomas, aneurysms, cholesteatomas, cysts and other tumors may be found in the cerebellopontine angle and may produce cochlear or vestibular symptoms; however, acoustic neurofibromas, which make up from 7 to 10 per cent of all brain tumors, are by far the most common lesion found in this region. This paper represents an analysis of 150 Mayo Clinic cases of unilateral acoustic neurofibroma in which an operation was performed and in which the diagnosis was confirmed microscopically. No attempt is made to list the complete neurologic findings in these cases since the purpose of this study is to evaluate the objective and subjective VIIIth cranial nerve findings suggestive of acoustic neurofibroma and to list the signs and symptoms, other than those resulting from decreased function of the VIIIth cranial nerve, which are commonly present in acoustic neurofibroma and which can be elicited with available equipment. When these are found, further neurologic evaluation is indicated.

Evaluation of deafness and tinnitus and other subjective symptoms in a series such as ours is difficult. Many of the patients were seriously ill or mentally confused and their ability to respond to questioning was limited. In other instances the recording of data left something to be desired; however, we feel that the following data are sufficiently accurate to serve the purpose of this presentation.

SIGNS AND SYMPTOMS OF DECREASED FUNCTION OF THE VIIITH CRANIAL NERVE.

Deafness or tinnitus or both were the initial symptoms in 105 of our 150 cases of acoustic neurofibroma (70 per cent). In most instances, as in Cushing's series, the impairment of hearing progressed steadily and rather rapidly to complete or nearly complete deafness (see Table 1). It will be noticed that in 85 instances the deafness was total and in 29 instances it was severe, though probably not total. Thus 114 (76 per cent) of our patients presented a marked hearing loss on the side of the neurofibroma. In 19 instances (13 per cent) the ipsilateral hearing loss was moderate. In one instance there was an islet hearing loss for the frequencies 2,048 and 4,096

and in another instance an ipsilateral hearing loss beyond the frequency of 4,096. It is interesting to note that the patient presenting the islet hearing loss had a nonfunctioning vestibular labyrinth on the side of the lesion, with normal horizontal and vertical canals on the contralateral side. The patient with hearing loss beyond the 4,096 frequency had a very hypoactive labyrinth on the side of the neurofibroma and normal horizontal and vertical canals on the opposite side.

TABLE 1.
SYMPTOMS RESULTING FROM DYSFUNCTION OF THE VIIIth NERVE IN 150 CASES OF ACOUSTIC NEUROFIBROMA.

Symptom	Number	Per Cent (of 150)
Hearing loss or tinnitus as first symptom	105	70
Total hearing loss on side of lesion	85	57
Severe but not total hearing loss on side of lesion	29	19
Moderate hearing loss on side of lesion	19	13
Bilateral hearing loss	6	4
Islet hearing loss on side of lesion	1	1
Hearing loss beyond 4,096 frequency	1	....
Tinnitus	100	67
Vertigo or questionable vertigo	16	11
Dizziness	53	35

The disproportion between decreased cochlear and vestibular function such as was present in these two cases should bring to mind the possibility of acoustic neurofibroma.

In six cases the hearing loss was bilateral and in two of these six cases the bilateral deafness was complete. Occasional contralateral hearing losses or findings indicative of disturbed function of other cranial nerves on the side opposite the acoustic neurofibroma are to be expected when it is remembered that these tumors sometimes produce marked torsion of the brain stem with consequent injury to structures in the contralateral cerebellopontine angle.

In the remaining 15 cases of our series of 150 there were insufficient data to permit evaluation of cochlear function. It is interesting to note that all adequately examined patients in

our series showed some degree of hearing loss, although other observers have reported verified instances of acoustic neurofibroma with normal hearing.

Tinnitus was present in 100 of our patients having acoustic neurofibroma (67 per cent). It was a prominent and frequently an initial symptom, but except for calling attention

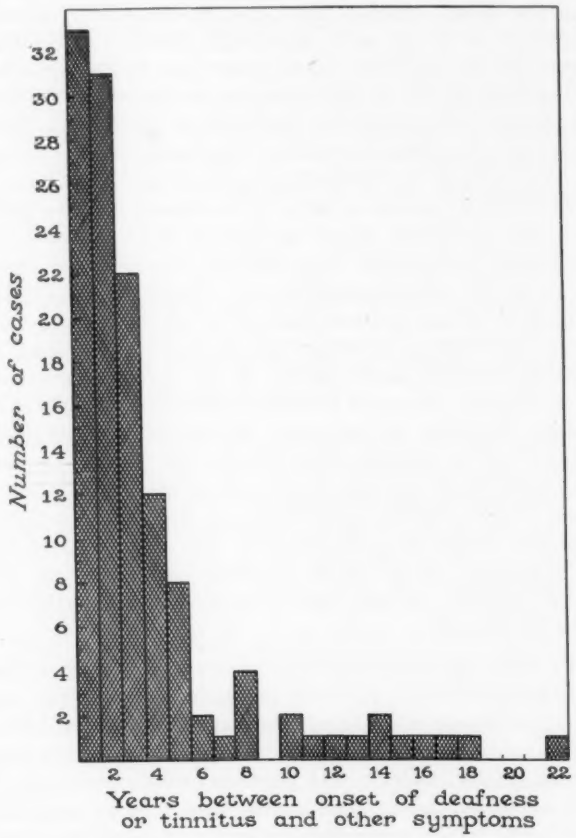

Fig. 1.

to the ear it offered nothing of diagnostic value. Almost every imaginable type of tinnitus was found in one or more of these patients; blowing, hissing, escaping steam, and pounding, synchronous with the heart beat, and constant or intermittent tinnitus were recorded. In some instances the tinnitus was present for months or years and then disappeared, and not infrequently deafness was present without tinnitus.

Since acoustic neurofibromas arise from the vestibular portion of the VIIIth cranial nerve, disturbed equilibrium might be expected to be an early symptom; however, presumably owing to the slow growth of the tumor and to the remarkable compensatory ability of the contralateral labyrinth and other mechanisms of body balance, imbalance of the type ordinarily encountered in dysfunction of the vestibular nerve was not a common symptom. In the unusual patient the symptoms may be confused with those of Ménière's disease, or the sudden onset of deafness with violent vertigo, such as was present in one of Cushing's patients, may simulate labyrinthine vascular accident, but in most instances real vertigo is absent; however, it must be remembered that patients with classic paroxysms of true vertigo accompanied by nausea, vomiting and fluctuating deafness and tinnitus do not present much of a problem and the diagnosis of typical Ménière's disease is easily made. Patients having some degree of unilateral nonfluctuating nerve deafness, with or without tinnitus, possibly with a prominent anxiety overlay and periodic attacks of imbalance which cannot be definitely catalogued, are difficult to evaluate. Many patients fall into this somewhat atypical group, and even though gross symptoms and signs of intracranial disease are absent, tumor of the cerebellopontine angle must be considered.

In our series 53 patients (35 per cent) complained of dizziness, lightheadedness, postural imbalance or similar equilibrial disturbances other than frank ataxia which we did not feel were sufficiently suggestive of Ménière's disease to cause confusion. In 16 instances (11 per cent) there was a history of true or questionably true vertiginous attacks associated with deafness which simulated end-organ disease; but in 13 of the 16 instances there were symptoms of an intracranial

lesion at the time of the onset of vertigo, leaving three out of 150 patients whose early symptoms might easily have been confused with those of Ménière's disease.

Of the three patients just mentioned, one was a 40-year-old man who had noticed a unilateral hearing loss for nine years and several attacks of true rotatory vertigo before numbness of the upper lip and poor vision brought him to the clinic.

The second patient, a 61-year-old woman, had had unilateral deafness without tinnitus for nine years and vertiginous attacks without nausea for six months before severe headache and paresthesia of the left side of the face caused her to seek medical aid.

The last of the three patients was a 48-year-old man who came to the clinic because of vertiginous attacks lasting for periods ranging from 15 minutes to three hours, which at times were sufficiently violent to cause him to fall. Unilateral deafness and tinnitus had been noticed for seven years. There was a history of numerous indefinite functional complaints of long duration to confuse the clinical picture. The objective findings were moderate nerve deafness on the left, nonfunctioning left vestibular labyrinth with normal right labyrinth, questionable left corneal hypalgesia and questionable mild ataxia. The ocular fundi and Roentgenograms of the head were normal. Tumor of the cerebellopontine angle was thought to be a possibility, but the findings were not considered definite enough to warrant surgical exploration. The patient returned to the clinic a month later. At that time the equilibrial disturbance was constant and was markedly increased by postural change, the ataxia and corneal anesthesia were definite, there was a first to second degree spontaneous nystagmus to the right and left, and there was some questionable weakness of the left side of the tongue. Surgical exploration demonstrated a left acoustic neurofibroma.

At the time the first and second patients just mentioned came to the clinic for examination, findings relative to the central nervous system were sufficiently evident for the proper diagnosis to be made, but had these patients appeared for evaluation several years earlier, undoubtedly neurologic signs would have been less pronounced, and the differentiation between damage to the end-organ of the VIIIth cranial nerve and acoustic neurofibroma would have been more difficult. Probably also a few of the 53 patients who complained of some type of imbalance other than real vertigo, and whose condition as disclosed in the review of the case records seemed so definitely not labyrinthine, would have presented more of a diagnostic problem had we seen them earlier when imbalance, deafness and decreased labyrinthine function were less overshadowed by other neurologic findings.

Moderate ipsilateral nerve deafness, nonfunctioning vestibular labyrinth on the side of the lesion, and normal horizontal and vertical canals on the opposite side were the objective findings with respect to the VIIIth cranial nerve in the third patient just referred to. This would suggest and logically it would seem, since acoustic neurofibromas originate from the vestibular portion of the VIIIth cranial nerve, that labyrinthine function is apt to be decreased earlier than is cochlear function. This is in agreement with Ireland's findings.[4] In an earlier paper, two of us (Brown, Love[5]) reported the results of caloric testing on 18 of this same group of patients who presented less hearing loss than would be expected in the presence of the usual acoustic neurofibroma. In every instance there was a nonfunctioning or a very underactive ipsilateral vestibular labyrinth. Application of the alternate binaural loudness balance test for recruitment introduced by Fowler[6] would probably have been useful in evaluating the condition of the patients with atypical hearing loss; this test is particularly applicable when there is doubt as to whether the deafness is of end-organ origin or is due to injury to the VIIIth cranial nerve. Dix, Hallpike and Hood[7] observed that recruitment of loudness was present in Ménière's disease but was absent in tumors of the cerebellopontine angle. Eby and Williams[8] obtained similar results in their series of patients.

In the paper by Brown and Love[5] it is reported that in 73 of 79 patients adequately examined, a nonfunctioning or a very hypoactive labyrinth was found on the side of the acoustic neurofibroma, with normally reacting horizontal and vertical canals on the contralateral side; in only six of 79 instances were nonfunction of the vertical canal and normal function of the horizontal canal on the contralateral side associated with nonfunction of the ipsilateral labyrinth.

The time of onset of decreased labyrinthine function in instances of acoustic neurofibroma usually cannot be accurately determined; however, hearing loss, particularly if accompanied by tinnitus, is troublesome to the patient and may lead to otologic consultation before other symptoms of the tumor develop. The interval between the onset of deafness

or tinnitus and the development of symptoms suggesting intracranial disease as the causative factor in the decreased cochlear function is of interest. In 27 per cent of 125 instances in which the history seemed fairly definite, deafness was the only symptom for one year or less; in 25 per cent evidence of intracranial disease appeared from one to two years after onset of initial hearing loss or tinnitus; and in 18 per cent deafness or tinnitus alone had been present from two to three years. Thus in 70 per cent of 125 patients, symptoms of acoustic neurofibroma not related to decreased function of the VIIIth cranial nerve developed within three years of the onset of the initial deafness or tinnitus. The longest interval between the onset of deafness or tinnitus and the appearance of other symptoms was 22 years. It is, therefore, apparent that, although intracranial evidence of acoustic neurofibroma usually appears within a few years after the first symptom, occasionally deafness or tinnitus alone may be present for a considerable number of years and that the presence of acoustic neurofibroma must be considered in almost any instance of unexplained nerve deafness with or without tinnitus.

SIGNS AND SYMPTOMS OTHER THAN THOSE OF DECREASED FUNCTION OF THE VIIITH CRANIAL NERVE.

Deafness, tinnitus and disturbed equilibrium are the symptoms of acoustic neurofibroma which are likely to result in otologic evaluation. Examination of the cochlea and equilibrial labyrinth produces evidence suggestive of the proper diagnosis, but this evidence must be linked with findings indicative of intracranial disease before the presence of acoustic neurofibroma can be suspected with reasonable certainty.

In Table 2 are listed the central nervous system signs and symptoms which, taken in conjunction with those of decreased function of the VIIIth cranial nerve, were indicative of acoustic neurofibroma in our series. Spontaneous nystagmus was present in 125 instances in our series (83 per cent). As Cushing pointed out in his series, the nystagmus was usually horizontal, present on looking both to the right and to the left, and more often coarser and slower toward the side

of the lesion. The quick and slow components of the nystagmus, although definitely present, are not so distinct as in labyrinthine nystagmus, and although in most instances they are not present when the patient is looking straight forward, they become rather wild and active on slight deviation of the eyes from the midline, either to the right or left. The nystagmus of the two eyes is commonly in perfect rhythm, and although it appears to be especially vigorous, it is easily reversed by caloric stimulation of the ear.

TABLE 2.

FINDINGS IN ADDITION TO DYSFUNCTION OF THE VIIIth NERVE THAT WERE SUGGESTIVE OF ACOUSTIC NEUROFIBROMA IN 150 CASES.

Symptom or Sign	Number	Per Cent (of 150)
Corneal anesthesia	126	84
Ipsilateral	110	73
Bilateral	16	11
Facial paresthesias	77	51
Headache	105	70
Facial weakness	55	37
Moderate	9	6
Complete	1	1
Spontaneous nystagmus	125	83
Choked disk	74	49
Facial pain	16	11
Visual disturbances	47	31
Positive Roentgenologic findings	64	43
Positive Romberg sign	106	71

Corneal anesthesia was present in 126 of our patients (84 per cent), and in 16 of the 126 the decrease in corneal sensitivity was bilateral.

Headache was a prominent symptom and frequently was the one that caused the patient to seek medical assistance. While the records indicated that it could affect any part of the head, most commonly it was located in the ipsilateral occipital region; it frequently came on at night or soon after arising from bed in the morning, and it was often intensified

by movement of the body or straining of any sort. The headache usually increased in frequency and severity from the time of onset and was often accompanied by vomiting and occasionally by dizziness.

Seventy-seven of the patients (51 per cent) listed paresthesias as a prominent symptom; numbness, tingling, cold feeling, wooden sensation, or burning was the usual description of the symptoms commonly located about the face, gums or tongue and rarely in the extremities. Once the abnormal sensation had developed it was usually constant with a tendency toward spreading to adjacent areas, although it occasionally fluctuated in intensity and in one instance was present for one year and then disappeared. In two instances the paresthesia was on the side of the face opposite the acoustic neurofibroma.

Facial weakness was found in 55 patients (37 per cent). In only one instance was the paralysis complete. Usually the decreased muscle strength was so slight that careful observation was necessary to ascertain the palsy. In view of the course of the facial nerve through the internal auditory meatus along with the auditory nerve, it is surprising that it is not seriously injured more frequently in acoustic neurofibroma.

Facial pain, although present in only 11 per cent of our patients, is nevertheless a symptom to be kept in mind, since one occasionally sees instances of atypical facial pain for which no specific causative factor can be found and which in some instances is sufficiently severe to cause the patient to neglect mentioning a less troublesome tinnitus or deafness unless he is directly questioned. Two of our patients had undergone dental extractions in an effort to obtain relief from the discomfort.

Choked disk was present in 49 per cent of our patients, and Roentgenograms of the head were helpful in the evaluation of 43 per cent.

SUMMARY AND CONCLUSIONS.

A review was made of 150 Mayo Clinic cases of unilateral acoustic neurofibroma in which an operation was performed and in which the diagnosis was confirmed by microscopic examination of tissue. Some degree of deafness was present in all cases. Commonly, the deafness was complete or severe, but occasionally it was mild or atypical. In all instances of mild or atypical deafness in which adequate caloric tests had been carried out, labyrinthine function was found to have been more severely depressed than cochlear function. The finding of nonfunctioning vestibular labyrinth on the side of the neurofibroma with retained activity of the vertical and horizontal semicircular canals of the opposite side was the usual result of caloric stimulation. On the basis of this study the following conclusions are drawn:

1. Any degree of nerve deafness with or without disturbed equilibrium in the presence of spontaneous nystagmus, decreased corneal sensitivity, atypical facial pain, paresthesias of the face or tongue (rarely the extremities), unexplained headache, facial weakness, visual disturbance or ataxia, is suggestive of an intracranial lesion and indicates that complete cochlear and labyrinthine evaluation should be carried out and that Roentgenologic examination of the head, examination of the ocular fundi and neurologic consultation are in order.

2. The finding of signs suggestive of intracranial disease on the side opposite that of the deafness does not rule out the possibility of acoustic neurofibroma.

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AN EVALUATION OF POSTURAL VERTIGO.*

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Postural vertigo may be defined as a disturbed sensation which occurs when the position of the body is altered with relation to the force of gravity. The exact mechanism by which it is produced is not known. It is known that the equilibrium controlling system includes the labyrinth, VIIIth nerve, brain stem, cerebellum, proprioceptive system and many other centers of the higher central nervous system. Many of these neurologic areas and their connections are as yet poorly understood, and are easily disturbed by a number of factors. Change of posture, which may include coincident changes in blood pressure, heart rate and, occasionally, respiration is often such a disturbing factor; however, what we now commonly call postural vertigo has come to mean vertigo accompanied by positional nystagmus, which occurs on changes of the body from one position to another.

It is impossible to discuss postural vertigo without speaking and thinking simultaneously of positional nystagmus because positional nystagmus is the objective sign which can be observed. For example, dizziness often occurs when a person rises suddenly from a sitting or lying position. This is a type of postural dizziness, and it is not accompanied by nystagmus when it is on the basis of a drop in blood pressure alone. Dizziness of this type may last several minutes, or it may persist as a feeling of lightheadedness for an hour or two. It is not violent, and it may be caused by postural hypotension, anemia, low blood sugar, or arteriosclerosis. While this is a postural dizziness by the definition given, we call it postural hypotension or transient syncope.

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When we speak of postural vertigo we mean a sudden occurrence of true whirling which usually lasts for a few seconds or, at most, a minute. It is much more violent than the sensation of postural dizziness. It may occur with any changes of body position, but most frequently it occurs when the patient lies down or looks up with the head tilted backward or when he turns from one side to the other while lying down. When this occurs there is no doubt in the patient's mind that some violent change has taken place. He feels he must hold onto something to keep from falling, even though he may be lying down. The whole room may suddenly seem to turn upside down. Usually he immediately changes back to the position from which he moved. Just as suddenly the vertigo disappears. There is no accompanying nausea. This violent vertigo does not occur every time he assumes the position which brought it on, but it most frequently occurs during a specific position change. In other words, if a patient experiences his vertigo on turning to the right, this is usually the only change in position which produces it for him, though it may not occur every time he assumes this position. In other patients vertigo may be brought on by other changes of position.

The symptom is severe enough so that the patient usually mentions it voluntarily; however, in any otoneurologic study a specific inquiry should be made as to its occurrence. To make sure it occurs, it must be demonstrated. One can usually tell that vertigo has occurred by the violence of the patient's reaction; but to prove that it has occurred, one must see the accompanying positional nystagmus. Positional nystagmus, therefore, becomes the diagnostic sign which establishes the presence of postural vertigo.

This type of nystagmus is demonstrated by positional tests. These are simple to perform and require no special equipment except Frenzl glasses, and these are not necessary because the nystagmus can be seen without them; however, the nystagmus is seen more frequently and more easily with Frenzl glasses. The technique of testing, as described by Lindsay and others, is as follows: The patient is seated on a table and is then

helped through a series of different positions: *1.* from sitting to supine, to sitting, then to supine again; *2.* from supine to the right and back to supine; *3.* from supine to the left and back to supine. The testing is concluded with the head-hanging position. This is carried out by either lowering the patient's head as the end of the table is dropped or by asking the patient to move up on the table so that his head hangs over the end.

When the patient lies down, sits up, and turns, care should be taken that the head and neck remain in the same relation to the body. Insofar as possible, this eliminates reflexes from the neck muscles and compression of the vertebral arteries. Experimentally, both have produced nystagmus. Many authors state that positional nystagmus is most easily demonstrated in the head-hanging position. Yet, this position allows the factors of stretching and compression to alter the reaction. Since this is true, the head-hanging position test should be done on a posture table which permits lowering of the head without disturbing its relation to the body. In partial review of the literature, I found only Nylén[10] emphasizing the use of a posture table. I must admit that I am among those who have been guilty of using the head-hanging test without considering that it might not be accurate. We have been taught to be careful to maintain the head and body relationship while going through the other position changes, yet we deliberately disturb this relationship by the head-hanging position and attach more importance to it than to the other positions.

Before we can evaluate postural vertigo and positional nystagmus, we must attempt to understand whether the nystagmus is spontaneous or induced. We are all agreed that spontaneous nystagmus indicates disease in the statokinetic system. We all know that induced nystagmus is an artificial sign by which we test the function of this system. I can find no criteria on which to base the assumption that positional nystagmus is a spontaneous reaction. Everyone agrees it is a reaction to a given stimulus, and that the stimulus is change of position. Spontaneous nystagmus is usually consistent in

its direction and it is sustained. Induced nystagmus is of limited duration and may be in any direction, depending upon the position of the body when the stimulus is introduced, whether the stimulus be ice water, hot water, or galvanic current.

If we are agreed that the foregoing is true, the positional nystagmus which accompanies postural vertigo is much more like induced than like spontaneous nystagmus. If this is true, it may not always indicate organic disease, even though we know it does not occur in the normal person.

Positional nystagmus which occurs together with postural vertigo may take several forms. It may occur in the same direction, it may be in opposite directions at different times, or it may be irregular. These are the three classical types of positional nystagmus described by Nylén as type I (direction changing), type II (direction fixed), and type III (transitory or irregular). Lindsay[8,9] uses this classification, as do most other workers in this field. It permits a standard nomenclature in discussing positional nystagmus. The only points of disagreement are in the interpretation and evaluation of what is indicated by each of the respective types.

The changes in positional nystagmus during inflammatory labyrinthitis, according to Nylén,[10] are:

1. Type II positional nystagmus toward the involved ear in early irritation of the labyrinth.

2. Spontaneous nystagmus toward the involved ear when the entire labyrinth is irritated.

3. Type I positional nystagmus when the utrical is upset.

4. Type II positional nystagmus toward the uninvolved ear (which he interprets as meaning paralysis of the utricular otolith).

5. Spontaneous nystagmus toward the good ear when the labyrinth is dead.

6. Clearing of the nystagmus when compensation takes place.

Gorman and Brock[6] stated that in posterior fossa tumors, parasites of the fourth ventricle, and following cautery of the midportion of the cerebellum of experimental animals, positional nystagmus occurs when the head is tilted backwards. Cogan[3] said that positional nystagmus is present in 80 per cent of posterior fossa tumors, but is never found in middle or anterior fossa tumors. Spaeth[12] was of the opinion that the side of the lesion is indicated by the direction of the quick component. Santamarina[11] studied 30 patients, 10 of whom had infratentorial lesions, 13, supratentorial lesions and seven, cerebellopontine angle tumors. He concluded that lesions of the central nervous system produce type I positional nystagmus, whereas disease of the peripheral labyrinth produces type II positional nystagmus; however, he pointed out that to localize disease it is also *necessary to find neurologic disease.* Clarke,[2] on the other hand, maintained that positional nystagmus is most commonly produced on extension of the neck, such as in looking up, and that it is caused by cerebellar tumors, disturbances of the otoliths, and injury of the head. Jongkees[7] recognized the three types of positional nystagmus and said it is generally accepted that types I and III are produced by lesions of the central nervous system, and type II by lesions of the peripheral labyrinth, but that type I may also be produced by the latter lesions. He destroyed the saccule in rabbits and came to the conclusion that damage of the peripheral labyrinth can cause: *1.* positional nystagmus, even when the caloric tests are normal; *2.* positional direction changing nystagmus (type I); *3.* vertical nystagmus.

Lindsay[8,9] observed that positional nystagmus is demonstrated infrequently in Ménière's disease. But Nylén[10] maintained it occurs commonly in Ménière's disease. He demonstrated it in 55 of 83 cases and pointed out that of more importance was the fact that in 13 of these there was type I (direction changing) positional nystagmus, most often towards the healthy ear. He mentioned, also, that in "typical" Ménière's disease there is either type II positional nystagmus

or no positional nystagmus, but that in questionable Ménière's syndrome there was type I positional nystagmus in 29 of 30 cases. He believed this may indicate that the cause of some cases of so-called Ménière's disease or syndrome originated from the central nervous system.

From the observations of these investigators, it is evident that a great deal of confusion still exists regarding the significance of postural vertigo and positional nystagmus. Certainly, the finding of positional nystagmus or postural vertigo or both without other symptoms or signs of disease can help us little. We all see patients who fall into this category. It is the patient who gives a history of sudden violent dizziness for a few minutes on turning over in bed. It may have occurred several times. He is alarmed. We question him, examine him, put him through the positional tests, do an audiogram and caloric test on him and find nothing. If we are persistent and re-examine him on several occasions, particularly as regards positional tests, we may be able to produce his vertigo and nystagmus once or twice and again obtain no further information.

What are we to tell the patient? Certainly from experience with these patients we know that they all seem to get well without treatment. In such cases, then, are we justified in assuming that there is central nervous system disease, even if the nystagmus produced is type I? Are we justified in assuming there is any disease? I for one am not sure of the interpretation. It appears to be more logical that we assume there is some disturbance in the proprioceptive reflex pathways which need not necessarily be in the brain or brain stem. In an excellent motion picture, Fowler recently demonstrated that damage to the neck muscles, eye muscles, the internal ear, and certain parts of the central nervous system produce a similar response in experimental animals. He also showed that in some patients whose equilibrium was already disturbed, a certain amount of stability could be attained by the use of prisms over the eyes. He did not say, nor does he know, that this means the disturbance of equilibrium is due to eye muscle disease; however, he was of the opinion that the

results are probably due to a dissociation of the reflex activity which goes on in this complicated system all the time. I am sure further work is being done all over the country as a result of Fowler's stimulating presentation.

Since we know, from experimental work, that specific injury to the otolithic membrane can produce positional nystagmus, that postural vertigo and positional nystagmus both occur in various dysfunctions of the central nervous system, and that there are no hard and fast rules by which we can interpret postural vertigo and positional nystagmus, I am convinced we are still in a dilemma. I do not believe that the demonstration of positional nystagmus and its accompanying vertigo has been of real value in making a diagnosis. It is my opinion that eliciting postural vertigo and positional nystagmus by positional tests merely indicates that a reaction has been induced in some portion of the statokinetic system. We do not know exactly where or why the reaction is produced. *In the absence of other information from the history or from examination and special tests, the finding of positional nystagmus and postural vertigo is of little value.*

We are not too concerned regarding treatment in cases of central nervous system tumor, Ménière's disease or acute labyrinthitis in which postural vertigo is a symptom. In these cases a diagnosis will be made and treatment will be instituted on the basis of history and other findings. The postural vertigo is an incidental symptom. Finding it does not establish the diagnosis nor does it influence the treatment; however, in the case in which there is a post-traumatic syndrome due to a head injury or in the case in which the only complaint is postural vertigo, some treatment beyond reassurance is desirable.

Fletcher[4] and others have pointed out that certain drugs, such as pyribenzamine and dramamine, and salt restriction therapy have been of no value. Clarke[2] is of the opinion that exercises designed to produce postural vertigo repeatedly in a given patient tend to diminish the postural vertigo. Cawthorne and Cooksey[1] demonstrated the value of exercises in

severe cases of postconcussion syndrome to re-educate the labyrinth. This is analogous to the education of the labyrinth which occurs in the ballet dancer or the skater who by constant whirling become able to remain free from dizziness after considerably more stimulation than is given to other persons. There are four basic exercises: *1.* repeated change from sitting to lying position; *2.* slow, then rapid head shaking; *3.* rotation while standing; *4.* throwing a ball to others standing in a circle. These exercises might well be added to our armamentarium since they have proved to be of value.

SUMMARY.

I have attempted evaluate some of the factors responsible for postural vertigo and have raised some questions which still need further answers:

1. Is the positional nystagmus associated with postural vertigo spontaneous or induced? It appears to be more like induced nystagmus.

2. Are we guilty of faulty technique when we use the head-hanging position as a reliable test for postural vertigo? There is some evidence that head-hanging introduces factors other than position alone and that these factors may alter the reaction.

3. Are the types of positional nystamus produced by testing actually helpful in localizing the area of the statokinetic system which is disturbed? As a corollary to this question, can we be sure that it always indicates disease? There are certainly differences of opinion.

4. Are we putting too much emphasis on a finding which is common but which without other signs and symptoms is of little value in diagnosis? I am inclined to believe we are.

I am aware that some of my views will be challenged, but it is with the hope that a difference of opinion will help clarify the problem that I have raised these questions.

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OTOLOGIC SYMPTOMS IN MULTIPLE SCLEROSIS.*

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Multiple sclerosis is one of the most confusing diseases with which physicians have to contend and it is proving just as baffling for men in the research and experimental fields. As there is so much confliction in the field of etiology, perhaps the safest manner to approach the subject is from the viewpoint of the pathologist. It is an acute or chronic, progressive or remittant disease involving chiefly the white matter of the central nervous system. During the acute phase there is swelling of the myelin sheath, followed by a destruction and loss of myelin and replacement by glial cells. Undoubtedly the swelling and absorption of myelin early in the process is of a reversible nature if the process has not been too rapid, and thus accounts for the relapsing nature which is so characteristic of this disease. The active process may terminate at any time during the evolution of the glial replacement, and so the involvement and interruption of the axis cylinder would vary in each lesion.

I would first like to show several slides of these sclerotic or glial areas of the brain and one in which a spinal nerve is involved. The usual site of these glial patches is in the white matter of the spinal cord and brain. The peripheral, cranial and spinal nerves are infrequently involved. It is particularly the phylogenetically younger structure, *viz.*, pyramidal tracts that are more apt to be first diseased.[1] An extremely common site is the vestibulocerebellorubral pathways. As this disease does not occur in animals, nor can it even be produced in animals, it may in some way be related to newly developed neural connections and myelinization. In this paper I have

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made an attempt to ascertain how often the auditory and vestibular tract of the VIIIth nerve are involved in a routine examination of known multiple sclerosis patients.

Statistically, multiple sclerosis is a relatively rare disease, occurring in the United States about 50 per 100,000 population. Thus by a slight margin multiple sclerosis outranks both intracranial tumors and neurosyphilis among the total number of admissions in a national hospital.[2] Its frequency is more significant when it is found that the disease is one of young people, having its onset almost exclusively between the ages of 20 and 40. The average age of cases in this series was 32—the youngest, 13, and the oldest 53. Graphs from a large series show much the same picture.

A very striking fact is that it is a disease of cool and damp climates. Although every state in the union is represented, it is several times more frequent in the northern states. Multiple sclerosis is relatively infrequent below the fortieth parallel, roughly a line drawn across the United States from San Francisco to New York City. The highest incidence on the North American continent is in British Columbia, the province immediately north of Washington and Montaina. The cause of the relation to temperature is unknown.

Although it is a serious disease, resulting many times in disability from the onset, the outlook is better than generally assumed.[3] A survey by Limburg indicates that the average duration of the disease in the United States is approximately 27 years. The mean age of onset is 28 years, and the average age at death about 54 years. On the basis of this survey it is seen that this illness lasts much longer than is commonly supposed.

Multiple sclerosis always seems to begin suddenly and with some dramatic event; that is, dramatic from the patient's viewpoint. Yet it is usually something which a physician is unable to define as a disease process. For example, a blurring of vision, yet no eye disease; a tinnitus or vertigo, yet no ear disease; a tingling and numbness of hand or foot, but no vascular lesion. As one talks to a group of these patients, he

gets an impression of a common factor in psychological make-up. It seems as though each individual has had a series of personal problems which he is not quite able to master. When such a patient presents his problem, with no evident pathological process, it is almost inevitable that he is labeled a psychoneurotic, and within a few days, or weeks, the double vision, scotoma, or, as in the first case to be presented, the facial palsy clears up. Frequently the recovery is complete, and both physician and patient are left confused as to the disease process or even whether a disease was present. Such is the usual onset of multiple sclerosis. Infrequently there is a massive fulminating illness with hemiparesis or blindness, and there are undoubtedly variations in both extent and locations of involvement to account for the many manifestations of the onset. In about 40 per cent of our series, the symptoms entirely disappeared for a few months or perhaps up to five years. In the remaining 60 per cent, the disease was progressive from the onset. For those cases in which the symptoms entirely disappeared, probably no diagnosis could be made, but with the recurrence of the symptoms or in the progressive cases, a second involvement, the diagnosis should have been made. It is here I feel that physicians as a whole and not infrequently the otolaryngologist should warn the patient of possible recurrence. It is at this point that the patient begins to make rounds of different doctors without receiving either a diagnosis or treatment. In this study a total of 50 cases were studied, with 28 able to give definite and accurate ENT histories. In the other cases histories were obscure and they were not included. For 20 of the 28 cases, ear and throat symptoms were prominent during the early manifestations of the disease. This represents about 65 per cent of the cases in a routine survey. In four of the cases the ear symptoms were the outstanding event at the onset. This represents about 2 per cent of all the patients. Of the group of cases showing symptoms relative to ear disease, the most frequent is that of vertigo, which occurred in 35 per cent of the cases. The next most common symptom was that of headache, which occurred in 25 per cent of the cases. Dysphagia in 20 per cent of the cases and tinnitus in 15 per cent of the cases were

the next most frequent symptoms. Of particular interest are two cases which show a moderately severe nerve deafness which appeared at the onset of the disease and which was followed by progressive sclerotic lesions in other parts of the brain. I feel that these represent actual involvement of either the auditory nerve or the cortex in the auditory radiation.

I do not mean to infer that ENT symptoms are the most frequent presenting symptoms of the disease; they are not. By far the most common are double vision, weakness or paralysis of an extremity. A peculiar and almost constant complaint I have never heard mentioned before in this disease is an extreme sensitivity to either cold or heat.

Even though a presumptive diagnosis of multiple sclerosis is made with the onset of some neurological lesion, there is almost no laboratory examination which will aid in its confirmation. By most accurate examination of the spinal fluid it is possible to determine a certain type of colloidal gold curve; however, this procedure is available only in large medical centers and the percentage of positive results is not high enough to make definite detection possible. Unless there are accompanying signs, such as a separate and discrete lesion of the central nervous system in some other than that of the primary involvement, the true nature of the disease will not be found. It must be said, however, that a careful neurologic test will usually establish whether or not a dysfunction is due to a single or multiple defect. The reason for emphasis on these particular facts is that successful management of a case depends upon early detection of the true nature of the disease. Once a scar has been formed, there is no reason to believe that there ever can be any function through that area. This also applies as in traumatic nerve injury.

Once a tentative diagnosis has been made, it is imperative that the following type of treatment be started. It is not with the feeling that a cure will be achieved, because at the present there is no cure for multiple sclerosis. Even though we realize this, there are certain approaches to therapy which bring immeasurable relief to the patient and credit to the medical

profession. These measures can be divided into three categories: *1.* drug therapy, *2.* psychotherapy, and *3.* general supportative measures. Drug therapy is directed mainly to those agents which may prevent the development of new lesions, *i.e.*, vasodilators. The most effective is histamine, whether given intravenously 2.75 mg. per 250 cc. daily, or by an iontophoresis method is immaterial. At present the most satisfactory method is by means of an electrical apparatus, made available by an electrical firm for a cost of only $7.50. In this method about 1 mg. of histamine is released over a period of several hours and can be used by the patient in his own home.

A more recent suggestion is the use of histamine in an oil suspension, which, like insulin, would be released over a period of 24 hours. This type of treatment should be continued daily and perhaps for the entire life of the individual.

The frequency with which I have heard patients say that the antihistamines, taken for an intercurrent respiratory infection, has precipitated a relapse is indeed striking. Even though there are no statistics on such occurrences, it would seem wise to ban this group of drugs.

Psychotherapy is perhaps one of the most difficult methods of treatment, but perhaps, after all, the most important. There is an increasingly growing acceptance of the idea that allergy plays a major rôle in etiology.[7] We have all seen urticaria resulting from emotional and food allergy. Having a common embryological origin with the skin, it may well be that the central nervous system responds with isolated areas of edema and pressure, with exposure to allergenic agents. With no method of expansion due to cranial confinement, necrosis follows. If the edema adsorbs, a remission occurs. Antiallergic treatment with histamine lends itself admirably to the promotion of other supportive measures. If these patients can be reassured and a program of morale building instituted, perhaps by a physician, but surely by some organized force, a great deal will already have been accomplished.

Among the general supportive measures, physiotherapy may aid in the prevention of contractures which prove so crippling in the later stages. Spasticity is one of the common late sequelae, as from any upper motor neuron involvement. The drugs which may relieve spasticity are mephanesin orally or curare in an oil suspension given intramuscularly.

SUMMARY.

Multiple sclerosis as a disease entity involves the brain stem in such a manner that vertigo, headache, tinnitus and hearing loss are not infrequent symptoms. These symptoms should be recognized when associated with other evidences of multiple sclerosis such as pyramidal tract or optic nerve lesions. The disease is a slowly progressing one and treatment with the above procedures should be instituted as early as possible.

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EXTRAMEDULLARY PLASMA CELL TUMORS OF THE NOSE, PHARYNX AND LARYNX: A CASE REPORT.*†

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This report presents a case of multiple plasma cell tumors of the nose, nasopharynx, oropharynx, hypopharynx and larynx (see Figs. 1, 2, 3, 4). Plasma cell tumors occur quite frequently in the bone marrow constituting one of the histologic types of multiple myeloma, according to Hellwig.[1] These tumors are adequately discussed in general textbooks, but the extramedullary tumors are usually reported as single cases, are much more rare and are less understood.

Hellwig,[1] in 1943, reviewed all the literature from 1905 to 1942. The present paper presents an additional case and reviews some of the literature for the period from 1942 to 1951.

C. S., aged 50, was first examined on March 15, 1948. The patient stated that 15 years before, he had had a benign tumor removed from the conjunctiva of the right eye and that about five years ago he had had his tonsils taken out and had some large polyps removed from his pharynx. He was seeking medical attention because he had difficulty in speaking and in swallowing.

Examination showed multiple round masses of tumor tissue protruding from the posterior choanae, the posterior pharyngeal wall and hanging from the soft palate (see Fig. 1). Indirect laryngoscopy showed a mass of tumor tissue completely blocking the view of the upper end of the larynx. The patient's dyspnea was remarkably slight. Biopsy was taken in the office, and the patient was found to have a plasmocytoma.

On April 22, 1948, the patient was admitted to Northwestern Hospital in Minneapolis where a thorough study for mulitple myeloma was made. Normal films of the ribs, spine, pelvis and long bones were obtained. On May 26, 1948, a preliminary tracheotomy was done under local anesthesia. General anesthesia was then induced and suspension laryngoscopy was carried out. A tumor mass 3 by 2 by 2 cm. was snared off of the right aryepiglottic fold, and two smaller tumors were removed from the

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vocal cords. Convalescence was satisfactory. The tracheotomy tube was removed five days after surgery, and the airway and voice remained good until October, 1948, when hoarseness recurred, but there was no dyspnea. On Dec. 27, 1948, a pedunculated pink tumor was removed from the larynx under general anesthesia with an intratracheal tube in place. The pedicle of the tumor arose above the cords, and the tumor hung down between the cords, causing hoarseness.

The patient got along well for one year. In December, 1949, he developed a fistula through his face opposite a lower molar tooth. This proved to be due to a dental abscess and not to tumor occurring in the mandible.

In May, 1950, one of the plasma cell tumors occurring in the right nasal space bled severely after the patient sneezed. Bleeding was controlled with oxycel gauze packed into the nasal space.

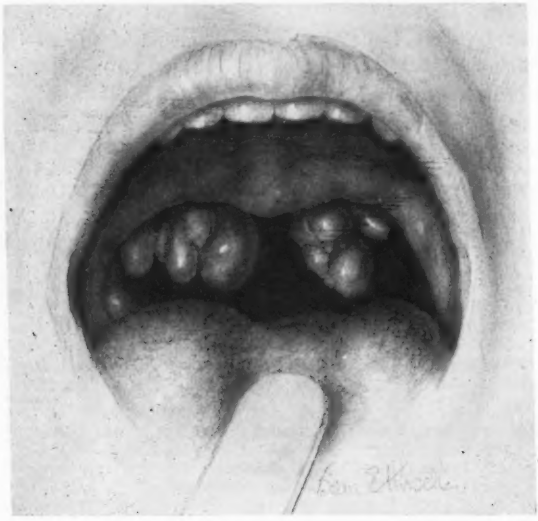

Fig. 1. Drawing of pharynx showing multiple plasmocytomas.

In April, 1951, a plasma cell tumor attached to the entire edge of the left vocal cord was excised by direct laryngoscopy. Numerous tumors were removed by blunt dissection from the nasopharynx and oropharynx at this time.

The patient has had no further trouble as of this writing (November, 1951) except for a nosebleed apparently caused by drying of his nasal tumor from dust inhaled at work. This was not a serious difficulty.

As previously noted, in 1943 Hellwig[1] reported on all articles which had appeared from 1905 to 1942. These comprised 127 published cases. In 63 of these, the tumor originated in the air passages; in 47 in the conjunctiva; in four in the

lymph nodes; and in 13 in other organs. It will be seen that the case reported in this paper includes the conjunctiva and the pharynx, which are the two most common sites.

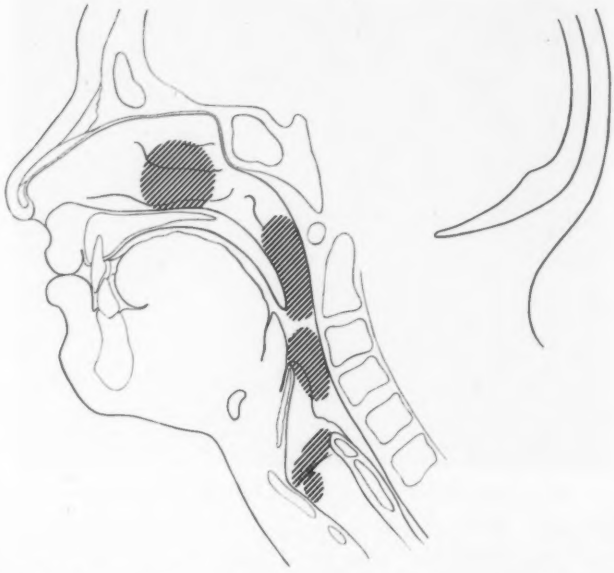

Fig. 2. Sagittal section showing locations of plasmocytomas in case reported here.

It seems from a review of published papers that plasma cell tumors occur as *a.* noncancerous single tumors, *b.* noncancerous multiple tumors (this seems to fit the case reported here), *c.* cancerous tumors without metastases, and *d.* cancerous tumors with metastases in the lymph nodes.

The case reported here seems to fall into the group of noncancerous multiple tumors. There were nine such cases reported by Hellwig.[1] The patients' ages varied from 46 to 70 years. The multiple tumors did not vary in their gross appearance from the solitary plasma cell tumors. They were lobulated or polypoid with distinct pedicles, and their consistency was firm. The color was described as pale gray and

sometimes dark brown or bluish red. In the present case this tumor was deep red in color. The microscopic picture was that of plasmocytoma. The plasma cells were the principal

Fig. 3. Low power view of plasma cell tumor of pharynx.

tumor cells. In the case reported here the pathologist commented that if he had seen the tissue without knowing the history he would have made an unequivocal diagnosis of multiple myeloma.

It seems that this man may ultimately develop metastases from his tumors, but he has gone for at least 15 years now and no one can predict whether he will continue for the rest of his life without metastases. From a therapeutic standpoint the fact seems to be established that as long as the plasma cell tumor is localized and confined to soft tissue, a clinical cure may be obtained by surgical removal or irradiation or both. On the other hand, therapeutic measures only rarely have prevented a fatal outcome if applied after the tumor had either locally invaded bone structures or had spread to lymph nodes or to the skeleton.[2] Cases are reported in which the primary tumor was excised and did not recur

for years and then secondary foci unexpectedly became manifest in lymph nodes, internal organs or bones and the disease progressed in a short time to an unfavorable outcome.

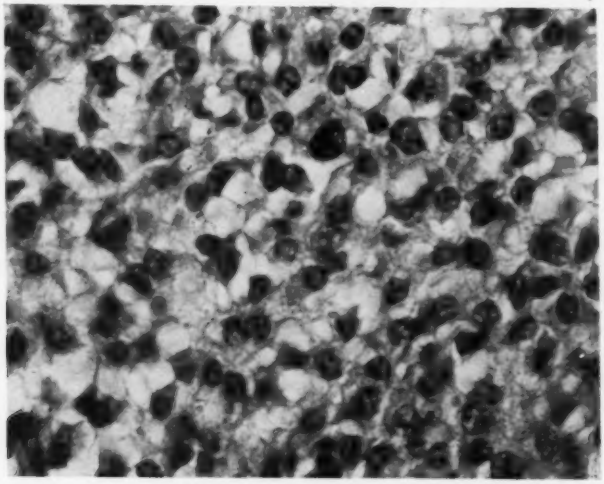

Fig. 4. High power view of same.

The physician is hard put at times to know the correct treatment in a case of plasmocytoma. Connor[6] says, "The inherent uncertainty of the prognosis substantiates the advice of Figi, Broders and Havens to consider plasmocytoma malignant and treat them as such." This advice seems cogent and surely should be applied to the easily accessible plasmocytoma. Figi[11] says, "In most instances symptoms have been noted for less than six months but they may have been present for a year or more. . . . In most of the cases the tumor appears clinically to be highly malignant, which is in keeping with the usual short history. At times, however, the growth presents a polypoid appearance. Early extension to the lymph nodes would naturally be expected in view of the activity of most of these tumors, but such involvement was present at the time of the primary examination in only one of the 11 cases in our series." Hellwig[1] says, "In 65 cases the tumor originated in the air passages; in 35 of these it was localized,

and in 30 it was definitely invasive." Hellwig[1] states further, "Wachter found five tumors in a 48-year-old woman — two in the nasal cavity, two in the nasopharynx and one in the larynx. . . . The patient reported by Wachter had only surgical removal of five tumors, and at the time of publication, 10 months later, three tumors had reappeared; however, 11 years after Wachter's report, Oppikofer found that this patient was still living and in good health 31 years after the onset of the disease."

Quoting Hellwig[1] still further, "The gloomy outlook of some authors, who regard extramedullary plasma cell tumors as fundamentally the same as multiple myeloma, is as unjustified as the opposite view that these tumors are, as a rule, noncancerous processes. If plasma cell tumors are observed for many years, it becomes apparent that as a rule they begin as localized lesions in mucous membranes. This clinically noncancerous phase may persist for a long time, sometimes for 10 years or more, but ultimately, without change in histologic character, the process tends to become generalized. Whether or not the dissemination, when it does occur, is the result of metastasis or of multicentric origin of the neoplasm cannot always be stated with certainty."

As Costen[10] says, "These are good examples of the paradoxical nature of every set of facts regarding plasmocytoma and multiple myeloma." Certainly the writer, charged as he is with the responsibility for applying safe and proper therapy to a patient most closely resembling the 31-year survival cited above, is faced with a difficult choice. Adequate surgical removal coupled with cauterization of the bases and possible use of radiation therapy for clinically benign tumors of the nose, nasopharynx, oropharynx, hypopharynx and larynx is a formidable undertaking; the failure to destroy only a few cells would invalidate the whole undertaking. If one were sure of stopping the whole process of tumor development the surgical exercise necessary would be justified; but in view of the possible "multicentric origin of the neoplasm," is this possible? Only time and the subsequent development of the disease can supply the solution to this medical conundrum.

SUMMARY.

A case of multiple benign plasmocytoma of the upper air passages is reviewed and the extremely difficult therapeutic problem is discussed.

ADDENDUM.

After this case report had been prepared a patient was examined and found to have a solitary smooth deep red tumor of his right pharyngeal wall. This tumor was about 3 cm. in diameter and had a broad base of attachment to the pharynx. It was not painful, and had been discovered accidentally by the patient's physician during an examination for another purpose. The tumor was excised under general intratracheal anesthesia and was found to have a histologic structure identical with the photomicrographs illustrated in this paper. Because this was a solitary tumor and having in mind Dr. Figi's comments quoted in this paper, the patient was treated with X-ray. The time is too short to know what will happen but it is thought desirable to get this case into the report since these are uncommon.

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CEPHALGIC MECHANISMS WITH SPECIAL REFERENCE TO THE ATYPICAL FACIAL NEURALGIAS.*

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Headache, colds and neuralgias[1] are common complaints that monopolize no small portion of time and effort in the field of otolaryngology.[2] They often are the symptomatic expression of vasomotor imbalance; a cephalic manifestation of the same derangement of the neurochemical control mechanism for autonomic functions, as abdominal migraine, peptic ulcer, mucous colitis, cardiospasm, angioneurotic edema, herpes zoster and asthmatic bronchitis.[3] Modern medicine envisages these diseases not only as the result of damage due to some pathogen but also as derangements of the general adaptation syndrome against nonspecific stress.[4]. Defense reaction against recurrent crisis often brings about such a marked degree of vasomotor, neuromotor, and secretomotor dysfunction in the target organs, especially in the upper respiratory passages and organs of deglutition, that minimal irritation from some local pathogen becomes adequate to serve as the trigger mechanism for one or other variety of cephalalgia.

Most men lead lives of quiet desperation. Periodic exacerbations of migraine, histamine cephalalgia, or psychoneurotic relaxation headaches follow intermittent periods of tension. These generalized headaches offer no special problem in diagnosis; neither do the specific cranial nerve neuralgias limited to the distribution of one or another main trunk of the trigeminal nerve, less commonly the glossopharyngeal nerve and rarely the nervus intermedius. It is realized, however, that

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in both entities, although the etiology of tic is as yet unknown, an underlying personality tension, not infrequently frustration, is common. It is generally believed that an underlying vasomotor imbalance is the common basic factor in all forms of headache.[5] This applies with special emphasis to the obscure deep seated atypical neuralgias within the head, behind the face and below the basis cranii. These neuralgias are much more common than tic douloureux; they affect people in all walks of life, especially in these days of atomic fission, insecurity and crisis.

Physicians themselves are not immune to the more or less crippling forms of cephalocervical myalgia. Cervical disc and other traumatic or congenital lesions; vertebral arthropathy; cervical fibromyositis and other low grade infections in the cephalocervical juncture have been treated—sometimes with less than brilliant results due to a lack of appreciation of etiological significance in concomitant derangements of the general adaptation syndrome.[4] Angioneurotic, vasospastic and musculospastic episodes that may be identified as part of the myalgic syndrome are periodic, polymorphic and not confined to the distribution of any one nerve. The pain is more or less continuous, deep seated, seldom of extreme intensity or paroxysmal.[5] Postherpetic neuralgia may approach that of tic douloureux in intensity, but the prognosis is good. The disease is self limited, although recurrences have been known.

Recurrent noninfected sore throat[3] is a nonspecific atypical myalgia that is representative of the catarrhal type of condition often referred to the otolaryngologist. No foci of infection may be uncovered; antibiotics are often ineffective for such conditions. A concomitant vasomotor rhinitis, however, along with mucosal edema and other evidences of vasomotor instability indicate ischemic myalgia of the pharyngeal constrictor muscle as the causative factor, *i.e.*, part of the alarm reaction in a derangement of the general adaptative syndrome.[4] Nonspecific emotional stress, occupational strain, domestic infelicity, frustration or fear may activate the hypophysis-hypothalamic neurochemical control mechanism more strongly toward increased adrenergic effects. Generalized

vasoconstriction, hemoconcentration, hypoglycemia and decreased permeability of the endothelial lining of the arteriole-capillary-venular units throughout the entire body are the effects of the alarm stage in the general adaptative syndrome. Vasospastic ischemia in the vasa vasorum and vasa nervosum may produce myalgia in striated muscle. Reflux secondary edema and vasodilation develop in the cholinergic reactive stage; tension upon perivascular nerve plexuses produces pain. It is in this reactive vasodilative stage of the intracranial blood vessels[7] that the hemicrania of migraine develops. Generalized cephalalgias due to psychoneurotic cause are probably produced in the same way. The local pain producing mechanism in the throat may or may not be associated with a pathogenic irritant that is subliminal by itself to initiate or prolong the disease. In the conscientious search for foci of infection, bacterial, viral, or antibody reagin H factors, oversight of some physical factor that may be superimposed upon the nonspecific defense reaction may fail to bring about permanent relief. The patient with an obscure neuralgia of the deep facial region or at the juncture of the head with the neck may become a medical orphan[5] who has been "massaged, analyzed, X-rayed, fitted with glasses," relieved of one or more turbinates and perhaps all of his teeth, and still emerges with headache intact.

Recent clinical experimental findings by Wolff[8] and his confreres cast a new light upon the sources and mechanisms for the production of pain in the human head. The pain sensitive structures are skin, mucous membranes,[9] basal dura mater, tentorium cerebelli, and the blood vessels both within as well as outside the cranial cavity. On the other hand, the skull, pia-arachnoid, brain substance, and the ventricles of the brain are insensitive to pain. The meninges of the vault are much less sensitive to pain than the basal dura; however, the middle meningeal (and other meningeal arteries) are pain sensitive, as well as the meningeal branches of the trigeminal, facial, glossopharyngeal, vagus, and upper two cervical nerves. Supratentorial intracranial irritation is referred to the superficial trigeminal area. Infratentorial meningeal irritation is

referred to the auricular, postauricular and suboccipital regions. Of all structures and mechanisms involved in the production of head pain, whether generalized or neuralgic, the most important factors aside from direct irritation to the pain carrying nerve fibres themselves, are the blood vessels.[8] Headache, according to Wolff,[10] is caused by the "irritation, traction or distension of a pain sensitive structure." The mechanism of headache, as in other types of *vascular pain*, is due to changes in the *vascular dynamics*. Alterations in blood volume and variations in the lumen of the blood vessels bring about irritation in the sensory nerve endings in vascular muscle as well as in the perivascular nerve plexuses.[11]

The pathways for transfer of pain stimuli from receptors to cerebral cortex are well established for the somasthetic system.[12] Exteroceptive nerves from the skin and bucconasal mucosa are general somatic sensory nerves. They follow the Vth, VIIth, IXth and Xth cranial nerves to their respective ganglia (trigeminal, geniculate, superior and jugular) and terminate in the *trigeminospinal* nucleus. Secondary somatic sensory neurones synapse with lower level spinal nerves, thus providing for referred pain to and from the neck. Ascending secondary somatic sensory neurones synapse in the thalamus. Tertiary sensory neurones carry the pain stimuli to the cortex. Thalamic reflexes and referral of pain have been described.[39] The cortex is in relation with cortical autonomic centers, and also with the hypothalamus. In this manner nonspecific stress factors, emotions and perhaps other reflexes may influence the hypothalamus, and indirectly affect the hypophysis and thereby modify the general adaptation syndrome.[4]

Pain originating in exteroceptor nerve endings is usually referred back to the site of origin. Muscles and tendons are supplied with proprioceptor nerves, about which all too little is known. The proprioceptor fibres of the muscles of mastication are unique in that their unipolar ganglion cells are not placed in the semilunar ganglion but in the mesencephalic nucleus[13] of the trigeminal nerve. It is generally agreed that all muscles are supplied with proprioceptor fibres, and al-

though the chief function is proprioception, under special circumstances such as ischemic myalgia, pain may be produced. The deep sensibility of the face, including the muscles of expression, is probably mediated by the facial nerve, and not exclusively by the trigeminal nerve.[12]

Enteroceptor nerves carry pain stimuli from the viscera. In the head there are two sets of general visceral afferent (pain carrying nerves).[14] These are located in certain cerebrospinal nerves; the VIIth, IXth and Xth cranial nerves, and in the perivascular nerve plexuses, that connect with the sympathetic trunk and the upper four thoracic spinal nerves.

The visceral sensory pain carrying nerves with which we are specially interested are accompanied (in large measure) by taste fibres, traverse the VIIth, IXth and Xth nerves to their ganglia (geniculate, petrous and nodosum) and all terminate in the nucleus solitarius.[15] Important reflex connections are established with the salivatory nuclei. Other connections are established with the somatic sensory nucleus and pathways previously described. The portions of these nerves in which we are especially interested are the glossopalatine nerve and the glossopharyngeal vagus cephalocervical nerve complex. It may be pointed out here that the embryonic pretrematic[16] branches of the glossopharyngeal (tympanic branch of Jacobson) and the glossopalatine nerve (great superficial petrosal, small superficial petrosal and chorda tympani) overlap in the territory that they supply. This region lies between the back of the face and the ear, between the basicranial axis and the visceral (gill) arches from which the jaws, palate, tongue, buccolaryngopharynx and pharyngotympanic tube are derived. It should also be pointed out that these nerves (together with the Vth) are evolved in the very earliest stage of embryonic development.[17] Their primary function is to supply the different branchial arches and their definitive organs. With the absorption of the dorsal portion of the gill arches into the basicranium in juxtaposition with the otic, ocular and nasal capsules, the primordial gill arch nerves (Vth, VIIth, IXth and Xth) not only become distributed to their own and adjacent gill arch derivatives, but also to the

sense organs and interior pain sensitive structures within the neurocranium. Meanwhile the primordial scleratogenous and cartilaginous neurocranium gradually undergoes ossification around the main trunks and branches of the gill arch nerves.[18] Congenital variations of excess or deficiency are not at all uncommon in zones of embryonic concrescence elsewhere in the body, and the juncture of the cartilaginous basicranium with the visceral skeleton is no exception to the general rule. Stenoses and dehiscences in the critical zone at the cephalocervical juncture between the basisphenoid and the maxillopalatine process anteriorly and the basisphenoid with its pterygoid process inferiorly (see Fig. 1) are commonplace findings in clinical anatomical laboratories.[21] The junctures between the basisphenoid and the alisphenoid (great wings) and between the sphenoid, basioccipital and temporal bones likewise show marked differences in the nature and degree of ossification in different skulls. In consequence of anomalous ossification, the different branches of the primordial gill arch nerves and their companion blood vessels often wind their way precariously through narrow hiatuses, canals or canaliculi, passageways with a lumen no larger than that which would accommodate a wheat straw.

This feature applies with special frequency to the different branches of the glossopalatine and glossopharyngeal vagus complex. Such coaxial cables are the motor secretory, and general and special afferent nerves, which, as branches of the VIIth and IXth, intermingle with each other in the same pattern that is characteristic of the cranial, cervical and sympathetic nerves elsewhere at the base of the skull. The small superficial petrosal is a good example of the plexiform intermingling of cranial autonomic nerves, and also of the variable patterns of transit through the skull. This small nerve formed within the petrous temporal bone by the union of VIIth and IXth pretrematic nerve fibres[16] has been traced through at least four different routes in its exit from the middle cranial fossa. These canals which should be mentioned briefly here vary from the usually large and complete foramen ovale, the foramen spinosum, to the inconstant canalis

innominatus, and the vestigial petrosquamous suture that also serves as a hiatus between the potentially infected middle ear and the middle cranial fossa. Other canals and canaliculi for branches of the VII, IXth and Xth nerves may now be listed briefly; the mastoid canaliculus for the auricular branch of the vagus (Arnold's nerve); the tympanic canaliculus (Jacobson's nerve); the hiatuses for the greater and

Fig. 1. Basis cranii externa. Anomalous lacerated foramina, and dorsal orifice of the pterygoid (vidian) canals; probes indicate course of vidian nerve; note the more cephalic position of the left vidian canal. The anterior orifice and passageway through the porta nasalis into the left nasal fossa was by duplicated foramina not evident in this exposure. The elongated and hypertrophied styloid processes, well shown in this view, are clinically significant factors in the vascular type of cephalocervical neuralgia.

lesser superficial petrosal nerves; the vidian canal, and the other canaliculi or foramina that lead away in all directions from the pterygopalatine fossa. It may be noted that the pterygoid process[18] and the definitive pharyngotympanic tube occupy the central key position between the deep dorsal aspect of the face, the pharynx and the ear. It is this critical region that is supplied not only by the visceral sensory pain pathways of the VIIth, IXth (and in small part by Xth) but also by a clinically important visceral afferent pathway later to be described.

Perivascular afferent pathways accompany all the arteries. This applies with special emphasis to the branches of the carotid artery, all of which have been shown to be pain sensitive structures. Vasospasm[7] of the deep branches of both carotid arteries as they traverse foramina at the base of the skull results in obscure atypical neuralgias that simulate other clinical entities.[3] Only a few examples need be mentioned here, since they are representative of a wide field in the clinical anatomy of the head and neck. Vasomotor occlusion Bell's palsy;[36] vasospastic occipital neuralgia of the occipital artery may simulate organic suboccipital disease, functional muscular imbalance due to eye strain or other disease in the orbitonasal region; vasospasm of the posterior auricular artery has been confused with mastoiditis, but it may be confused with the referred pain over the vestigial lateral line nerves[20] (VIIth, IXth and Xth) in meningeal irritation in the posterior cranial fossa; vasospasm of the anterior auricular and anterior tympanic branches of the internal maxillary artery may simulate otitis externa, otitis media or Costen's syndrome; vasospasm of the infraorbital and alveolar branches of the internal maxillary artery may simulate antral empyema and dental disease; vasospasm of the ascending pharyngeal is a common cause of pharyngeal myalgia; the histamine cephalgia of the superficial temporal artery is a well known entity, but the deep muscular branches of the internal maxillary artery offer a field of clinical functional anatomy as yet to be developed.[3]

Although numerous other examples of obscure neuralgia due to vasomotor imbalance in the general cervicocephalic vascular mechanism could and perhaps should be given here, the scope of this article requires that we limit ourselves to the critical pterygoid, pterygopalatine, nasopharyngeal and pharyngotympanic zone. Recently an important visceral pain pathway has been established which transfers pain stimuli from the orbitonasal region to the dorsal root ganglia of the upper four thoracic spinal nerves. The route follows the deep petrosal nerve through the vidian canal, where, because of dehiscences in the roof of the canal, the nerve is exposed to

infection within the sphenoid sinus.[21] Not infrequently the nerve traverses the floor of the sinus immediately below the mucoperiosteum (see Fig. 2).

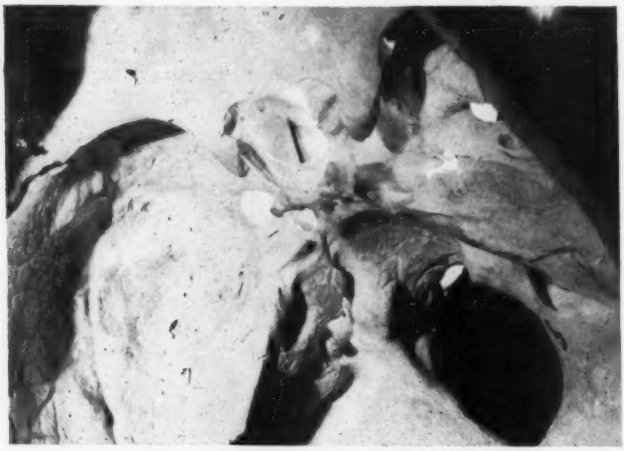

Fig. 2. Basis cranii interna. The middle cranial fossa of the same skull, giving a view of the floor of the left sphenoid sinus through a window produced by removing, in part, the floor of the sella turcica. The specimen was exposed to light from both above and below in order to reveal the marked translucency of the floor of the sphenoid sinus and the course of the vidian nerve through the lumen of the sinus. There is no dehiscence in the right vidian canal, but the thin floor of the left sphenoid sinus extends across the midline to the medial margin of the right foramen ovale. The left sinus also extends dorsally to occupy the entire basisphenoid and anteriorly encroaches upon both optic canals. The left sphenoid sinus during life was separated by thin bone from both optic nerves, both cavernous sinuses, the contents of both Meckel's caves, basilar venous sinuses and other neurovascular structures lying upon the clivus; as well as the pterygopalatine fossa and the roof of both nasal fossae. The right sphenoid sinus is present, placed in contact, but separated from the most posterior ethmoidal cell by a thin lamina of bone. The sphenoidal septum is almost transverse and attached laterally at the margin of the right optic canal. Both sphenoidal sinuses open into the sphenoethmoidal recess on their ipsilateral side. Although the illustration does not give a direct view of all these relations, they may be visualized from the extensive illumination through the floor of the sphenoid sinus.

As the deep petrosal nerve descends through the foramen lacerum it is subject to compression in a juncture of embryonic concrescence that is notorious for incomplete and irregular ossification (see Fig. 1). The pathway then follows the sympathetic plexus of the internal carotid artery and along the sympathetic gangliated trunk, without, of course, any synapse within the sympathetic system; finally, the pathway attains

the dorsal root ganglia of the uppermost thoracic spinal nerves via the white rami communicans. Severance of the thoracic nerve roots and Marchi staining technique by Christensen[19] has established the origin of the vascular visceral pain pathway in question, to be in the nasociliary and other nasal nerves. Cervicothoracic sympathectomy produces not only profound cholinergic effects within the nasal mucosa,[22] as may be expected, but also relief of pain in the orbitonasal region.

Gradually, year by year, the functional anatomy and pathology of the cephalocervical juncture has been clarified. The pioneer commentators, Sluder,[23] Vail,[24] Ramsay Hunt,[25] Glaser,[26] Fay,[27] Costen[28], and others who have contributed much to this field were handicapped by incomplete knowledge of the basic neural anatomy. Some misconceptions still remain concerning the functional anatomy of the vidian nerve (nerve of the pterygoid canal) and the sphenopalatine ganglion. In common with other cranial autonomic ganglia it is an important parasympathetic-motor ganglion, which mediates cholinergic effects over a wide terrain that radiates in all directions from the pterygopalatine fossa. The sphenopalatine ganglion, however, contains no sensory cells and no sympathetic (adrenergic) cells. Strictly speaking, the terms "sensory root" and "sympathetic root" are misnomers, and since they are misleading should be deleted from anatomical description of the sphenopalatine and other cranial autonomic ganglia. Since the sphenopalatine ganglion makes no synaptic connections with any sensory nerve, its etiological relationship in the domain of so-called sphenopalatine neuralgia is evidently limited to its cholinergic activity. The mechanism of the deep seated neuralgias in the orbitonasal region and the heterogeneous "sphenopalatine pain" involves differential diagnosis between those factors that would stimulate the more acutely pain conscious somasthetic mechanism versus those that have been grouped under the general adaptation syndrome.

Although the controversy concerning the sphenopalatine ganglion[30] and the status of sphenopalatine neuralgia and vidian nerve neuralgia[31] is still being waged in the current

medical literature[32] there is hope that the newer concepts of functional pathology based upon more accurate clinical anatomy of this region will clarify the problem. There is still some doubt as to the constancy of pain pathways in the pretrematic branches of the glossopalatine and glossopharyngeal nerves.[30] The great superficial petrosal nerve apparently functions chiefly for motor secretory effects in the orbit and nasopalatine regions. Gustatory and general afferent pathways are quantitatively less well represented; however, in the light of our knowledge concerning the intermingling of nerve pathways, and the establishment of the vascular pathway as an important clinical entity,[19] the vidian nerve (nerve of the pterygoid canal) should not be entirely written off as an important source of deep, obscure neuralgia. The geniculate ganglion is another important question mark. Does it or does it not contain the cell bodies of cholinergic neurones that supply the intracranial arteries?[15] Up to now, the geniculate ganglion, in terms of neural anatomy, has been accepted as containing only sensory ganglion cells, *viz.*, those belonging to the cutaneous branch of the VIIth, and the visceral sensory cells for taste and general sensation. The recently published work of Costen[33] and his confreres confirms the long prevalent impression concerning the functional components of the chorda tympani nerve. This important pretrematic branch of the facial nerve[16] that supplies the mandibular embryonic process contains not only cholinergic or motor secretory fibres to the salivary glands and taste fibres from the tongue, but also general afferent nerves that mediate the stimuli not only for touch and temperature but also pain fibres as well.

The purpose of this article is to appraise the clinical anatomy of the rather common atypical neuralgias at the juncture of the head and neck. In the light of recent evidence concerning the general mechanism for the production of head pain and the relative importance of pathogen versus defense,[4] our purpose will have been accomplished if it advances the viewpoint of functional rather than morphological pathology based exclusively upon morbid anatomy,[34] in the differential diagnosis of atypical deep facial neuralgia. Visualization of

basic causative factors provides the essential indications for treatment, whether antibiotic, antihistaminic, desensitization,[35] adrenergic, cholinergic or surgical. Whichever method of treatment, or combination of modalities is employed, the more promptly that decongestion of the pain sensitive structure can be accomplished, the better the end-results.

The lesions of the facial nerve[36] as it traverses the canal from geniculate bend to exit exemplify the differential diagnosis and management of atypical facial neuralgia. The common denominator of the different varieties of peripheral facial paralysis (Bell's palsy) is vasomotor dyscrasia. Whether the cause is traumatic (skull fracture or operative), infectious (focal infection from upper respiratory infections, mastoiditis, or teeth), allergic (as in vasospastic occlusion) or neurotrophic (herpetic geniculitis)—some form of circulatory deficiency is at fault. The pathogenesis is either traumatic or functional ischemia, with secondary edema and/or vasodilation. Severe pain is manifested in the vascular occlusion type (postauricular) and in the neurotrophic geniculitis (deep auricular). The distribution of the herpes zoster,[37] characteristic for Hunt's syndrome, is diagnostic when present (ear, palate, fauces and anterior two-thirds of the tongue). In the nonherpetic form (herpes sine eruptione) differentiation from tic of the pars intermedius VIIth can be made by anesthetizing the external auditory meatal "trigger zone." The trigger effect is thereby eliminated in tic, similar to the same technique in diagnosis of glossopharyngeal tic.

Obviously the modality of treatment varies in all the above cases, but the sooner that decongestive methods are employed the better will be the results, especially in the traumatic and vascular occlusive cases. The scope of our paper does not permit elaboration of the management of either motor disabilities of tic douloureux. One additional example of the many deep seated neuralgias of the cephalocervical region, while classed as atypical, is so common and the findings so pathognomonic and, furthermore, the results of operative treatment so uniformly satisfactory, that it deserves special attention. An elongated hypertrophied styloid process not uncommonly

compresses the perivascular nerve plexus of the external carotid artery, and produces pain in the region of the tonsillar fossa that simulates glossopharyngeal neuralgia. Differential diagnosis is aided by the X-ray findings; operative removal of the hypertrophied styloid process gives uniformly satisfactory relief.[38]

Addendum: Although intracranial neurosurgical procedures are beyond the scope of this article, mesencephalothalamotomy[39] has been successfully employed in a case of atypical facial neuralgia for which a prior retroganglionic neurectomy had failed to control the pain.

SUMMARY AND CONCLUSIONS.

1. The deep seated atypical neuralgias at the juncture of the face, basicranium, nasopharynx and pharyngotympanic tube have been appraised in the light of recent evidence establishing the location of pain sensitive structures in the head and neck, the pathways for transfer and referral of pain stimuli, and the general mechanism for the production of pain in the head and neck.

2. The present status of emphasis upon functional clinical anatomy and functional pathology, rather than upon morbid anatomy, in modern medicine and in the field of otolaryngology, has been considered especially as applied to the diagnosis and management of the common derangements of the general adaptation syndrome that culminate in cephalalgia.

3. Some of the factors of basic importance in the evolution and development of the upper respiratory passages, the organs of deglutition and the ear have been presented in order to elucidate the cephalgic mechanism wherein a pathogen, in this instance a congenital anomaly, may serve as the specific irritant or trigger mechanism, although subliminal in potency except at intermittent periods.

4. The diagnosis and management of atypical neuralgias met with in otolaryngology is in accord with the modern conception of disease which is viewed not only as the reaction to

a specific pathogen, but also from the viewpoint of defense against nonspecific stress and strain in some derangement of the general adaptation syndrome.

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CLINICAL OBSERVATIONS ON THE USE OF AUTOGENOUS VACCINE MADE FROM CULTURES OF COAGULASE POSITIVE HEMOLYTIC STAPHYLOCOCCUS AUREUS.*

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In considering the problem of immunization with autogenous vaccine we are well aware of the alternate enthusiasm and despair that has accompanied this form of therapy in the past. It is not our intention to describe a procedure to be applied indiscriminately to all cases of respiratory infection or that in any way relates to the so-called "common cold." It is the sole purpose of this paper to present the results to date of immunization with an autogenous vaccine prepared from young cultures of coagulase positive hemolytic staphylococcus aureus. Interest in this procedure was reawakened by results of a similar program conducted by Dr. David H. Scott and Miss Anita B. Mangiaracine, the co-author of this paper,[1] of the staff of the Massachusetts Eye and Ear Infirmary. This prior study was conducted by the ophthalmological service, and as I have based a great deal of my work on their results I quote verbatim from their report:

"With the advent of the sulfonamides, a new era in treatment of infections was begun. Within the past 15 years we have seen the emphasis on treatment shift from biological preparations to chemical preparations; however, there are still an appreciable number of people with infections that are not controlled by any of the currently available chemical drugs. In ophthalmology, one of the most perplexing problems we encounter today is in just such a situation — chronic

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infections of the eyelids, conjunctiva, or cornea. In other words, chronic external infections of the eye. In such cases we are again forced to return to the biologicals in the hope of finding some relief for the patient.

"In the experience of the bacteriology laboratory at the Massachusetts Eye and Ear Infirmary, the most common pathogenic bacterium isolated from chronic inflammation of the eyelids, conjunctiva, or cornea is the coagulase positive hemolytic staphylococcus aureus. This coincides with the observations published by Thygeson,[2] as well as those of O'Brien and Allen.[3] In many of our cases, cultures of the conjunctiva have been negative, but nasal cultures showed the organism.

"The ability of certain staphylococci to coagulate citrated blood plasma was first described by Loeb[4] in 1904. Production of coagulase, as this property has been termed, is considered one of the best criteria to pathogenicity. In 1944, Field and Smith[5] reviewed the status of various tests for pathogenicity and ended by concluding that, since the production of coagulase has better than 96 per cent correlation to pathogenicity, this property should be the main criterion for division of staphylococci into two groups: *1.* pathogenic (coagulase producers), and *2.* saprophytic (produce no coagulase).

"The toxin-producing properties of staphylococci have been extensively studied; and the situation was well summarized by Rigdon[6] in 1940. The toxin elaborated by pathogenic staphylococci has been divided into at least five fractions: lethal, dermonecrotizing, hemolysin, leucocidin, and enterotoxin. The first (lethal) may be shown by injecting a filtrate into laboratory animals, which develop a chill followed by fever and die within a few hours. The second is demonstrated by intradermal injection into rabbits, which produces an area of erythema that is followed by necrosis and sloughing. Hemolysin is divided into two fractions: alpha and beta. Alpha hemolysin is demonstrated by the ability to lyse rabbit red blood cells *in vitro* and sheep red blood cells less efficiently. Beta hemolysin hemolyses sheep cells better than it does those of other animals. Leucocidin tends to lyse white blood cor-

puscles. Enterotoxin produces a severe gastrointestinal disturbance in man. The production of toxin *in vitro*, according to Rigdon, reaches its maximum in an optimum medium in two or three days; and there may be some deterioration in media incubated for several days.

"Staphylococci have usually been considered to be non-encapsulated; but in young (three-hour) rapidly-growing cultures capsules have been demonstrated. Lyons[7] considers the capsular antigen to be of utmost importance in preparing autogenous vaccines for the treatment of staphylcoccus infections and states that some of the toxoid preparations which have been found to give clinical improvement have shown the presence of capsular antigen also. Fleming[8] has said that in his experience vaccine works in some cases and toxoid in others; and that sometimes one works where the other fails.

"In 1944, it was the general impression among the staff members at the Massachusetts Eye and Ear Infirmary that their results with the use of commercially prepared toxoid were not entirely satisfactory. One of us (A. B. M.), who had previously been associated with Lyons, suggested that an autogenous vaccine prepared from young (three to four-hour) cultures might be of benefit in these stubborn ocular infections. This seemed to offer the possibility of help, since previous failures with vaccine preparations described in the literature had been with old cultures — and often washed and resuspended in saline.

"Prior to November, 1948, this specially prepared autogenous vaccine had been given to a total of 78 patients. With the cooperation of all staff members who had used this material on private patients, as well as the records on clinic patients, we have been able to obtain some data on 71, with follow-up of three months or longer on 52.

"The course of treatment as given is outlined on an instruction sheet, which is given to the patient, who then carries it with him; and as each injection is given, it is checked by the physician, and any untoward reactions are noted so that subsequent doses may be adjusted accordingly.

MASSACHUSETTS EYE AND EAR INFIRMARY.

date:

The outline given below has been found to give excellent results when used to give the staphylococcus vaccine prepared from young cultures and used without washing the organisms. This vaccine is dispensed from the laboratory in the undiluted form and must be diluted as necessary by the physician giving it.

1st week		
1st day	Dilute vaccine in saline 1/100	inject 0.2 cc. i.m.
2nd day	Repeat	
3rd day	Repeat	
Rest 4 days		
2nd week		
1st day	Dilute vaccine in saline 1/100	inject 0.4 cc. i.m.
2nd day	Dilute vaccine in saline 1/100	inject 0.8 cc. i.m.
3rd day	Dilute vaccine in saline 1/10	inject 0.2 cc. i.m.
Rest 4 days		
3rd week		
1st day	Dilute vaccine in saline 1/10	inject 0.2 cc. i.m.
2nd day	Dilute vaccine in saline 1/10	inject 0.4 cc. i.m.
3rd day	Dilute vaccine in saline 1/10	inject 0.8 cc. i.m.
Rest 4 days		
4th week		
1st day	Undiluted vaccine	inject 0.2 cc.
2nd day	Undiluted vaccine	inject 0.3 cc.
3rd day	Undiluted vaccine	inject 0.4 cc.
Rest 4 days		
5th week		
1st day	Undiluted vaccine	inject 0.5 cc.
2nd day	Undiluted vaccine	inject 0.75 cc.
3rd day	Undiluted vaccine	inject 1.0 cc.
Rest 4 days		

6th week, 7th week and 8th week

For these three weeks give 1.0 cc. undiluted vaccine on the three successive days.

The above is only a suggested regimen to be used in giving the vaccine. It may be changed by the physician giving it to suit the needs of the patient. A. B. Mangiaracine, Bacteriologist.

"These tables are based on results as observed at the completion of treatment in the 71 patients on whom we have that information. Fifty-one of these showed improvement and 20 were unimproved. Seventeen were called "cured" or "markedly improved," but two of these relapsed. Of the 51 improved, we have been able to get at least a three months' follow-up on 45. Fourteen out of this group had relapses (some going as long as two years before relapses occurred). Among the 32 with no known relapse, 12 have been followed longer than a year.

"Among the 20 who were unimproved, we have no further information on nine, 11 have been observed three months or longer after completing the vaccine, and had not shown any improvement that could in any way be construed as anything more than the fluctuations in the condition as observed in the natural history of the disease."

In view of the fact that definitely beneficial results were obtained in ophthalmological infections caused by this organism, it seemed to the authors that this procedure might offer some additional help in the control of staphylococcal infections of the upper respiratory tract. We have all had our disappointments in the control of recurrent nasopharyngitis and sinusitis even after we have performed technically satisfactory operations on the tonsils and adenoids or on the sinuses. Likewise, antibiotics and chemotherapeutic drugs, however successful for the relief of the current infection, have left us with a very important problem. We can eliminate any number of pathogenic bacteria from the respiratory tract and elsewhere by the use of these drugs, but having done so the patient's immunity may be no greater than before the onset of the disease; in fact, I suspect that because we have combatted his infection artificially with drugs, he may have produced fewer antibodies with which to combat a subsequent attack should he be unfortunate enough to meet with the same pathogenic organism again.

One of these organisms, coagulase positive hemolytic staphylococcus aureus, is so prevalent in the nose and throat that

some have suggested that its presence is a normal biological condition. Yet it is capable of great injury when the balance between its virulence and the patient's immunity is upset in its favor.

The cases presented are selected from my own private patients. The bacteriological studies and preparation of the vaccines have been done by Miss Anita B. Mangiaracine, the bacteriologist at the Massachusetts Eye and Ear Infirmary, who has also collaborated in the preparation of this paper. As you will see in the charts, it is not a large group numerically as compared with some of the studies conducted in a quest for a cure for the "common cold." I have selected this group, hand picked them, for one purpose. They all present a problem of immunity. Operations had previously been done where indicated; a few required reoperation; in some no indication existed for surgery. Some had responded to antibiotics or chemotherapy or to allergy treatment, but there still remained the problem of building resistance to staphylococcal reinfection.

CHART I — AGE.

By Decade	No. Cases
1st	2
2nd	10
3rd	9
4th	10
5th	2
6th	6
7th	3

69 per cent between ages 10-40

In this series of 42 cases the ages ranged from six to 65; a greater majority, 69 per cent, were between the ages of 10 and 40. This was to be expected in view of the fact that most of the patients were old enough to have had surgery and medical treatment in the form of antibiotics and chemotherapy previous to vaccine; also, this age bracket includes the more strenuous and active years of one's life where great pressures are brought to bear on the individual while at

school and college, and in the earlier years where he establishes himself professionally or in business, or as in the case of a housewife where her family responsibilities are particularly exhausting. In other words, these people were subjected to much physical and emotional strain which contributed to a lowered resistance to infection.

As you will see in Chart II, the greatest number of cases presented some degree of sinus infection. The 12 cases of recurrent, acute sinusitis present also a problem of chronic sinusitis. Even though the X-rays and physical examination between attacks did not always reveal significant pathology, nevertheless their sinuses seemed to succumb to repeated infection so consistently that one would suspect that there was residual infection which lasted over from one attack to the next; in other words, a chronic factor. Eleven cases that presented acute sinusitis with allergic manifestations also would have some degree of chronicity in the background, although the chief complaint was of an acute nature, and the same also can be said of the cases in which the disease was chiefly in the pharynx and nasopharynx; however, the four classifications in the chart correspond fairly accurately to the conditions presented in this problem.

CHART II.

Disease	No. Cases
1. Recurrent acute sinusitis	12
2. Sinusitis with allergy	11
3. Recurrent acute pharyngitis	17
4. Nasal furunculosis	2
Total	42

In Chart III, I have listed the types of operation which have been performed; some by myself and some by other surgeons previously. Five had had no surgery and seemed to require none; these can be said to have only a low-antibody level as their problem. T and A was the most commonly performed operation; sinuses have been operated on either as

the only operation or in addition to T and A in 11 cases; five cases required reoperation, one on the antra and four secondary T and A.

CHART III — PREVIOUS SURGERY.

A. Operations	No. Cases
1. None	5
2. T and A only	22
3. Sinus only	3
4. Nasal polypi	3
5. T and A and SMR	1
6. T and A and sinus	8
	42
B. Reoperation	
1. T and A	4
2. Sinus	1

88 per cent had previous surgery

In evaluating the results I have grouped the cases in the following manner: first, cases with no recurrences, either to the present time or to the time when I last heard from the patient. As you will see in Chart IV, there are seven of these cases; the duration of their improvement varies from three months to four years; the patient listed as of three months'

CHART IV — CASES WITH NO RECURRENCES.

Case	Disease	Duration of Improvement
C. B.	Pharyngitis	4 yrs. to present
J. DeN.	Furunculosis	3 mos. to last observation
J. C.	Sinusitis with allergy	3 yrs. to present
M. A.	Pharyngitis	18 mos. to present
F. D., Jr.	Pharyngitis	3 yrs. to present
P. McW.	Furunculosis	18 mos. to last observation
S. K.	Pharyngitis	1 yr. to last observation
Total, 7		Summary:
		3 mos. 1
		1 yr. to last obs. 3
		3 yrs. 2
		4 yrs. 1

improvement left this country at the expiration of that time, so that I have no way of telling whether he is still improved.

In Chart V, I have grouped 12 patients who have coagulase positive staphylococcus aureus still present in recultures; these patients however, are grouped together because they definitely had fewer and less severe infections when they did occur during the time that they have been under observation.

CHART V — FEWER OR LESS SEVERE INFECTIONS ALTHOUGH ORGANISM IS STILL PRESENT IN RECULTURE.

Case	Disease	Duration of Observation
N. S.	Sinusitis	4 mos.
R. W.	Pharyngitis	4 mos.
M. P.	Sinusitis: Allergy	3 mos. to present date
R. W.	Pharyngitis	3 mos. to present date
C. C.	Pharyngitis	3 mos. to present date
M. S.	Sinusitis	3 mos. to present date
M. G.	Pharyngitis	1 yr. to present date
R. G.	Pharyngitis	2 yrs. to present date
J. L.	Sinusitis	1 yr. to present date
G. G.	Sinusitis	2 yrs. to present date
T. T.	Pharyngitis	17 mos. to present date
P. R.	Sinusitis: Allergy	2 yrs. to present date
Total, 12		

In Chart VI are six cases in which there was a very definite history of allergy with superimposed infection. Three of these cases were under active treatment for pollen hay fever,

CHART VI — IMPROVEMENT IN ALLERGY WITH SUPERIMPOSED INFECTION.

Case	Disease	Duration of Observation
K. S.	Pollen allergy and sinusitis	2 mos.
A. M.	Pollen allergy and sinusitis	3 mos. to present date
P. B.	Pollen allergy and sinusitis	9 mos.
W. S.	Allergic sinusitis with infection	9 mos. to present date
B.F.	Allergic sinusitis with infection	3 mos.
A. B.	Allergic sinusitis with infection	3 mos.
Total, 6		

and three presented infection of the sinuses as the chief etiology in their allergy; these cases have been under observation for only a short time compared to the previous groups that I have mentioned. To this group might be added two cases in the group to follow, but I have listed these two elsewhere for another reason.

In Chart VII, cultures were repeatedly negative after the course of treatment for an appreciable length of time; there are 12 of these cases, which include the two cases of sinusitis with allergy. In this group observations have been made at periods from three months to three years. As you will see in the summary, there are four who have been observed from three to nine months; five up to 14 months; and three up to three years.

CHART VII — NEGATIVE CULTURES AFTER TREATMENT.

Case	Disease	Duration of Observation
R. S.	Sinusitis	6 mos. to present date
R. McC.	Pharyngitis	3 mos. (last observation)
M. D.	Sinusitis	3 yrs. to present date
D. A.	Sinusitis	1 yr.: then recurrence
F. T.	Sinusitis: Allergy	1 yr. to present date
J. B.	Pharyngitis	3 yrs.
P.M.	Pharyngitis	14 mos.
R. E.	Sinusitis	1 yr.: then recurrence
P. D.	Pharyngitis	1 yr.
J. G.	Pharyngitis	9 mos.
P. L.	Sinusitis: Allergy	5 mos. to present date
W. K.	Pharyngitis	3 yrs.
Total, 12		Summary
		3 mos.- 9 mos. 4
		9 mos.-14 mos. 5
		3 yrs. 3

In the next chart are the five cases in which there was no improvement. These cases are still under observation, and three of them are still continuing their courses of treatment.

Finally, in the last chart I have attempted to compare my own series with those of Dr. Scott and Miss Mangiaracine in

CHART VIII — NO IMPROVEMENT.

Case	Disease	Observation
F. P.	Sinusitis	2 mos.
A. C-J.	Pharyngitis	3 mos.
D. H.	Allergy; sinusitis	3 mos.
T. C.	Pharyngitis	9 mos.
G. W.	Sinusitis	2 yrs.
Total, 5		

the previous study on the Eye Service. Under the eye listing, "improved," I have included those cases of my own in which there were no recurrences or in which there were negative cultures subsequently. In the eye series are those listed as improved but with subsequent relapses; this corresponds fairly closely with my group of fewer or less severe infections; there are 14 in the eye series and 20 in my own. Under "no improvement," 20 in the eye series and five in my own. To

CHART IX.

Summary	Eye Series S. and M.		N.-T. Series G. and M.	
Improved (no recurrences)	37		17	
Improved; then relapse (fewer or less severe)	14		20	
		34		25
No improvement	20		5	
Total	71		42	

compare those with maximum improvement in the two series with those with either relative improvement or none, in other words, to draw an absolute line somewhere in the picture, there is a relatively higher percentage in the eye series than in my own; but combining both series there were 54 improved as against 59 relatively or not at all improved. This would be the most conservative comparison of the two groups; but if one is looking for definite improvement even though it is not 100 per cent, one could say that out of the 113 cases in the combined series, 88, or 78 per cent, have been definitely benefited by this form of treatment.

In conclusion I reiterate that vaccine therapy is not a substitute for other recognized forms of treatment, either surgical or medical or just good hygiene. It has no relationship whatsoever to the problem of the common cold. It relates to allergy only insofar as the allergy is caused or aggravated by this form of staphylococcal infection; but where lower resistance to staphylococcal infection is a key factor in the management of the patient, this method of treatment with this vaccine has been of distinct benefit.

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GLOMUS JUGULARE TUMORS.

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Recently some writers have reported on the occurrence of a tumor of the middle ear and mastoid variously known as glomus jugulare tumor and/or nonchromaffin paraganglioma. The recognition and segregation of this tumor entity of the middle ear followed only belatedly upon the discovery by Krause,[9] in 1879, and the rediscovery, in 1941, by Guild[7] of a structure in the petrous portion of the temporal bone histologically resembling the carotid body and located just above the dome of the jugular bulb and/or in close association with the tympanic nerve of Jacobson and the inferior tympanic branch of the ascending pharyngeal artery. It was not until 1945, when Rosenwasser[17] found the first carotid body-like tumor in the middle ear and mastoid and suggested its origin from the glomus jugulare, that the possibilities of such growths became evident.

Although tumors of the middle ear and mastoid answering this description were previously encountered by competent observers, the correct diagnosis and pathogenesis was unrecognized due to the previous erroneous classification of angioma or angioendothelioma generally given to these growths. In a recent survey of tumors of the middle ear and mastoid by Mattick and Mattick,[14] the present writers became aware of two such erroneous diagnoses in a series of 10 cases of middle ear and mastoid tumors reported from here. Since then we have culled from our files a third case, which, on account of a pulsating mass in the mastoido-occipital region, had been mistakingly diagnosed some years ago as an arteriovenous aneurysm. A short history and findings of these three cases follows:

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Case 1: Female, age 17, admitted April 1, 1942. Family history not remarkable. Past history generally negative. Present history: Noticed deafness commencing in left ear in Fall of 1941 on routine school examination. About a month before admission she was referred to a specialist who noted a growth in the left ear canal and removed a section which was diagnosed as showing a general inflammatory picture, but in some areas showing strands of questionably malignant epithelial cells in fibrous connective tissue. She was referred here. There was no history of any pain or discharge from the ear.

Physical examination showed a 17-year-old girl who was well developed and who did not appear ill. The examination otherwise was not remarkable except for the left ear canal, where a mammilated, congested appearance of the integument covering the posterior superior portion of the ear canal and the posterior superior quarter of the tympanic membrane was noted. The anterior portion appeared quite normal. The nasopharynx was negative except for some adenoid remnants. Tuning fork test showed lateralization to the left and slightly prolonged bone conduction on this side together with moderate deafness. There was no tenderness or swelling over the mastoid and no cervical adenopathy. Section taken from the growth under topical cocaine showed papilloma. Wassermann was negative. X-ray films of mastoid and petrous region showed good aeration of cells and no evidence of osteolytic or hypertrophic bony changes. Patient was admitted to the hospital May 23, 1942, for insertion of a special radium tube in the left ear canal against the lesion. The tube was left in 15 hours (5 mgm. x 15 hours) after having removed another section for biopsy, which was now reported as an aural papilloma. In June there was considerable shrinkage of the tumor in the posterior canal and drum region of the left ear. There were no nodes. Lateral mastoid films on June 10, 1942, showed slightly less aeration of cells in the left mastoid area but were negative for any osteolytic changes. Nov. 27, 1942: Considerable telangiectasis from the radium application in the left ear canal and posterior half of the drum was noted. The Eustachian tube on this side was inflated. July, 1943, landmarks on left tympanic membrane were quite easily visualized. The long process of the malleus was clearly defined, but the posterior portion of the drum and adjoining canal wall was still thickened and reddened. There was no evident tumor but moderate conductive deafness in this ear. Weber lateralized to this side and the Rinne was negative. Patient was working daily. A year later, July, 1944, the posterior inferior quadrant of the left tympanic membrane and skin of the adjoining ear canal was red and slightly scarred. Patient was working daily and had considerable hearing in that ear. No discharge or pain. Oct. 1, 1945, patient returned, stating that since her last visit she had married and given birth to a normal child. During this pregnancy, however, she had developed a left facial paralysis coming on insidiously and included the left eyelids and corner of the mouth. Examination of the left ear canal showed a recurrent tumor mass covered by reddened integument apparently attached to the inferior posterior portion of the drum and extending over the adjoining posterior ear canal wall. There were dilated vessels in the surface of this tumor mass which almost occluded a view of the drum, which on last visit had been well visualized. X-ray films of mastoid showed increased cloudiness and decreased aeration of left mastoid region but no evidence of osteolytic bone changes. P. A. chest film was negative. The tumor mass was aspirated under topical cocaine on the assumption that it was cystic and 1 cc. of bloody fluid of no diagnostic import was obtained. A radical mastoid operation done under ether anesthesia Dec. 29, 1945, through a posterior aural incision. Antrum was

opened and bridge taken down. Tympanic membrane, malleus and incus were removed. Tumor mass was located in the middle ear, arising from the posterior inferior part of annulus tympanicus and apparently extending into posterior ear canal wall. The tumor shelled out quite readily. Skin flaps from posterior ear canal wall were sutured to deep fascia overlying temporal muscle, cavity packed with iodoform gauze and drained through ear canal. Posterior aural incision was closed with interrupted dermal. Histological diagnosis of angiofibroma was made here, and that of hemangioendothelioma of the middle ear was made by U. S. Army Otolaryngologic Tumor Registry.

Postoperative course was uneventful, facial paralysis remained unchanged, and the ear canal was dry. Jan. 16, 1946, a radium tube was constructed for fractioned treatment giving 250 mgm. hours on Jan. 16 to Feb. 2, 1946, with 10 mgm. radium element in gold 1 mm. On Feb. 23, 1946, there was no recurrence of tumor, no nodes noted. Facial paralysis unimproved. Pateint referred for X-ray therapy—8,400 r., 200 K.V. to left ear and mastoid Feb. 23 to March 5, 1946. No recurrence or nodes since.

Case 1 Histopathology: Tumor as seen in photomicrograph (see Plate 1) was made mostly of vascular channels distended with red cells. Large strands of collagen ran through the specimen. Between these forms of stroma were seen many epithelioid cell nests arranged in alveoli but

Photomicrograph showing histopathology as in Case 1.

many seemingly lying free in the interstices of vasculary channels. Microscopically, this tumor is more vascular and the picture less characteristic than that of the carotid body which it often closely resembles.

Case 2: White married female, housewife, age 57, on admission Nov. 2, 1942. Family history not remarkable. Past history: Para 2. Broke wrist in 1937. In 1930 noted pounding in left ear and bloody discharge. Ear was lanced. Had been losing hearing in left ear over past 30 years. Present illness: Spring, 1942, noticed growth in left ear, which was described as polyp which bled profusely. Section taken prior to admission was diagnosed here as xanthomatous polyp. Had four X-ray treatments to the left ear before admission. Physical examination here showed a well nourished middle aged female, fully edentulous and wearing plates. Heart and lungs negative, spleen not palpable, cervix lacerated. Pelvis otherwise not remarkable. Breasts not remarkable. Varicose veins on both legs. Reflexes not remarkable. Blood Wassermann negative.

Tumor obstructing ear canal was noted in the left ear. It appeared soft, cystic and pulsated. Skin over it was intact. Drum could not be visualized. Patient lateralized tuning fork to left ear; had negative Rinne, and was moderately hard of hearing on this side.

X-rays of mastoids showed slight cloudiness of left mastoid as compared to right—otherwise not remarkable. Submentovertex films generally negative. Patient refused operation, so radium bomb was applied to left ear canal tumor—4 mc/hrs. being applied q. two days for three treatments, making a total of 12 mc/hrs. in all, in four days, a very generous skin dose.

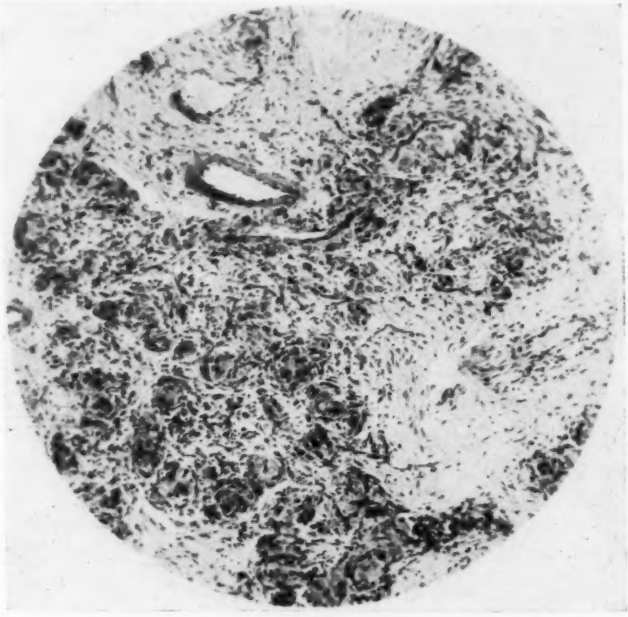
Dec. 10, 1942, mass in left ear canal was smaller. Aspiration of the mass yielded blood—ear otherwise dry. Since then patient has been ambulatory, feels quite well and steadfastly maintains that her ear does not trouble her and that she does not want operation. X-ray films at least twice yearly show cloudiness in left mastoid with poor cell definition—otherwise not remarkable. Patient was seen approximately every six months. Tumor in left ear canal was getting slightly larger but otherwise giving no more inconvenience. Patient still refuses operation. Oct. 19, 1949, patient had earache and some trismus, ear commenced to discharge. This was followed by pain. There was no mastoid tenderness. Films of mastoid were not remarkable. The tumor mass appeared red and pulsated with some discharge of pus and blood from the canal. Biopsy at this time was reported as ulcer. Silver nitrate was applied to ulcerous area, following which pus and blood ceased and the area epithelized over. April, 1950, left ear canal displayed a larger pulsating skin-covered tumor which obstructs the left external ear canal and a slight covered tumor which obstructs the left external ear canal. Tuning fork still lateralized to left with Rinne negative. Ear canal dry; no pain. X-ray of mastoid as before. Patient insists she feels well and wants no operative interference.

Case 2 histopathology: This tumor as seen in photomicrograph (see Plate 2) is made up of nests of epithelioid cells surrounded by collagenous fibrous strands between which are numerous vessels filled with red cells. In most places these cell nests form alveoli. These cells are mostly of the large, polyhedral type. An occasional multinucleated giant cell is noted. There is very close resemblance between this tumor architecture and that of the normal glomus jugulare.

Case 3: A white female, age 53 years, referred here June 4, 1943, for a pulsating tumor in and under the left ear.

Family history is not remarkable. Past history: had measles and typhoid as a child; influenza in 1915; four normal pregnancies between 1915 and 1927; tumor in left ear removed in 1929; and hysterectomy in 1930.

Present history goes back to discharging left ear while pregnant in 1927. Had tumors removed from left ear canal in 1929 and also had tonsils burned out, which stopped the discharge from left ear. In 1936, she noticed a "bunch" behind the left ear. Other than diminished hearing in this ear, she had no inconvenience from this mass. While having eyes examined, her doctor discovered tumor in left ear canal and a mass behind the ear. He took a biopsy, which was reported as malignant neoplasm, and she was referred here.

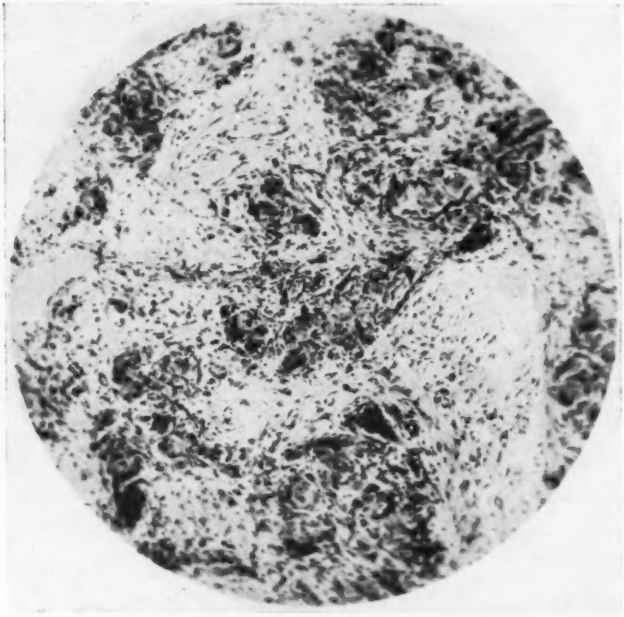


Photomicrograph showing histopathology as in Case 2.

Physical examination displayed a well nourished middle aged white female complaining of a left-sided headache and a constant pulsating, throbbing noise in the left side of her head. There was no real pain, but some purulent discharge from the left ear canal and some residual tumor tissue which blocked the deep external canal. There was apparent complete peripheral left facial paralysis and a visible swelling under the left ear, through which could be felt two confluent, soft subcutaneous

masses below the left ear canal posteriorly and under the left mastoid and occipital bone. This mass was soft and pulsated and apparently was the cause of throbbing noise in the head. She was moderately hard of hearing in left ear. There were no palpable nodes. Heart, lungs, abdomen and extremities were not remarkable. Two scars from previous operations on abdomen and moderate cystocele and rectocele were noted.

X-ray films of the mastoid showed decreased aeration of the cells on the left but no evident coalescence or destruction; there was apparent erosion of bone just posterior to the petrosa, which the Roentgenologist interpreted as suggestive of tumor involvement. Chest film was negative. Treatment: 200 k.v. X-ray therapy was applied to left mastoid and occipital region (7,400 r.), Aug. 14, 1943. This apparently had no noticeable effect. She commenced to notice dizziness and a tendency to fall forward. Left ear canal was still obstructed by tumor mass, which also pulsated



Photomicrograph showing histopathology as in Case 3.

and apparently was connected with the infra-aural and posterior mastoid larger pulsating mass, which was looked upon as an arteriovenous type of aneurysm. A bruit could be heard over the tumor mass. Patient was admitted to the hospital and operated upon on Feb. 16, 1944, under avertin and infiltration novocaine through an incision over the anterior border of the sternomastoid, extending up to the mastoid. The carotid sheath

was exposed and a large distended internal jugular presented in the wound. This was resected between double ligatures of chromic gut; and the underlying external carotid was ligated in continuity above the superior thyroid. The mass under the ear canal and mastoid was so vascular that no attempt was made to resect it, especially since ligation of the external carotid had apparently caused cessation of the pulsations. The deep fascia were closed and the skin sutured with silk. Patient made an uneventful recovery, was apparently free of the pulsating, throbbing noises and headache. The facial paralysis still persisted. The left ear canal was obstructed in its deeper portion, but there were no granulations or discharge. Hearing in this ear was still diminished; and a slight pulsation in the left upper cervical region under the mastoid could be felt on palpation.

The patient has been followed yearly to date. The obstructive swelling in the left ear canal has gradually receded until now the tympanic area can be visualized as red, fibrotic and devoid of the usual landmarks. The left ear canal is dry, the left facial paralysis persists. Patient has had no return of pulsating, throbbing head noise and no pulsation can be felt in the region of the former mass. There is still diminished hearing in the left ear, otherwise the patient is very well.

Case 3 histopathology: This tumor (see Plate 3) is more cellular and is characterized by more densely packed cell nests, in many of which the alveolar structure is difficult to discern. In some areas these cell nests of polyhedral epithelioid cells are surrouonded by cavernous network of dilated vessels packed with red cells. In other areas the vascular interstitial net is almost absent. There are a few areas where fairly large masses of collagenous material is deposited between the cell nest masses.

DISCUSSION.

It would seem that in the past the term endothelioma has been inadvertently and too freely used in an attempt to diagnose the less common tumors of difficult classification. Some of these lesions so diagnosed often ran a benign course and so belied such diagnosis; *i.e.*, endothelioma of the bronchus, which now is known to be the bronchial adenoma; the endothelioma of the pleura, now generally recognized as a metastatic pleural manifestation of a primary bronchus cancer; and last but not least, such lesions of the middle ear or mastoid of which there are a goodly number, approximately 33 to date, which are now reclassified as glomus tumors of otologic origin.

Marx,[15] in 1926, called attention to such controversial lesions in the middle ear. Ewing[4] long ago stated "on analysis it appears that various tumors which, at one time or another have led authors to employ the term 'Endothelioma' fall readily into other groups, and that this term has a very limited application."

According to Guild,[7] the organ which he named the glomus jugulare is a minute one with carotid or aortic body-like structure found in the adventitia of the dome of the jugulare bulb of the temporal bone and lies beneath the floor of the middle ear or in close association with the ramus tympanicus of the glossopharyngeal nerve of Jacobson. It usually measures 0.5 by 0.25 mm., may be multicentric, and, therefore, had previously escaped attention. It has the same innervation and blood supply, *i.e.*, the glossopharyngeal nerve and the inferior tympanic branch of the ascending pharyngeal artery, as the carotid body which in histologic structure it closely resembles. It is presently postulated by Boyd[1] and Lattes[11] that these bodies, consisting of nests of epithelial cells in a highly vascular stroma lined by endothelial cells, form a system of nonchromaffin paraganglia located along the course of the cranial nerves and vessels of the primitive branchial arches. They have been shown to have an afferent sensory innervation, probably parasympathetic; to secrete no hormone but to function most probably as chemoreceptors for regulation of blood pH. Tumors of these structures, generally benign and slow growing, are now looked upon as occurring in this system of nonchromaffin paraganglionic structures variously located in the orbit, mastoid bone and/or jugular bulb, nodose ganglion of the vagus nerve, carotid body; above the aorta as the organs of Zuckerkandl or supracardiac bodies, and in the pelvis as the glomus coccygeum.

Winship, Klopp and Jenkins,[18] in 1948, in a report of two glomus jugulare tumors describe in one what appears to be a malignant metastasizing type which involved an upper jugular node. Lattes[11] has reported the first such human tumors occurring in the aortic bodies. Lattes and Waltner[10] noted another glomus jugulare tumor which on autopsy metastasized to the liver. Dockerty, Love and Patton[3] reported a nonchromaffin paraganglioma of the middle ear with clinical symptoms of nystagmus, total left-sided deafness and facial paralysis which had previously been tentatively diagnosed as a cerebellopontine angle tumor. In the various locations above mentioned, all the tumors seem to have essentially the same

histological architecture, namely, epithelioid cell nests surrounded by a stroma of endothelial lined vessels interspersed with collagen strands. These tumors of the middle ear and mastoid are generally slow growing insidious affairs, producing but few symptoms and then only late in their course, *i.e.*, tinnitus, conductive deafness, and discharge due to painless tumor obstructing the ear canal. They are generally very vascular, bleeding easily and often profoundly on biopsy or operation. Later they locally erode the surrounding bone in their growth, often encroaching on the facial nerve and producing peripheral paralysis of this structure. Our Case 2, however, has had the tumor for many years without causing sufficient untoward symptoms to convince the patient of the necessity for operation. On the other hand, Case 1 showed a comparatively early involvement of this nerve by bony erosion due to its accelerated growth following pregnancy. Case 3 had apparently eroded through the mastoid and presented a pulsating tumor in the mastoido-occipital region, for which the patient sought help. The chief symptoms here were pounding noise in the head and facial paralysis.

Primary tumors of the middle ear and mastoid are generally conceded to be rare, ranging from two in 4,000 cases as noted by Furstenberg[5] at the University Hospital in Ann Arbor; six in 45,000 as reported by Junod[8] at the University Ear Clinic at Basel; five in 212,000 admissions at the Manhattan Eye, Ear and Throat Hospital in New York as seen by Robinson,[16] and approximately one in 7,000 suspected malignancies as reported from the Roswell Park Memorial Institute by Mattick and Mattick.[14]

Marx[15] collected 155 cases of primary tumor of the middle ear and mastoid distributed according to pathological diagnoses as follows: carcinoma, 55 per cent; sarcomas, various types, 35 per cent, and endotheliomas or hemangioendotheliomas, 10 per cent. On the other hand, we observed three such glomus tumors in 11 cases of middle ear tumorous involvement.

Winship,[18] *et al.*, have given a tabular list of 11 proven cases of glomus jugulare tumors recorded in the literature,

together with a similar list of 13 cases of probable glomus tumors, many of which had previously been diagnosed as endothelioma. In addition, several other reported cases dating back to 1913 are cited by them as very probably of this same origin. Recently Lewis and Grant[12] have reported two cases observed at the Memorial Hospital in New York.

From the literature it would seem that there is a great similarity in these tumors both clinically and pathologically. They occur with a greater relative frequency in females approximately in a three to one ratio and most often between 40 and 50 years of age, although in accordance with our Case 1 children may not be exempt. Lungren[13] has recently reported four cases and an additional nine cases which have been described under other pathologic designations, often endothelioma. More recently DeLisa[2] has reported a case in a 64-year-old woman, of approximately six years' duration with many local recurrences, but, like our second case, persistently refusing radical operation.

Similarly there is general consensus on therapy of these tumors, namely, radical mastoidectomy as an approach and means of removal of the tumor, as experience under the former diagnoses of angioendothelioma would seem to indicate. There seems to be no reason for radiation of glomus tumors as presumably, like carotid-body tumors, they should be radioresistant; however, Kurt Graf[6] in a report of eight cases suggests favorable response to deep X-ray therapy. Although all three of our cases had irradiation as an initial treatment, we are not inclined to depend on it exclusively or recommend it as the sole means of therapy. The prognosis under such management has generally been good for the glomus jugulare tumors more recently reported and also many of the so-called "angioendotheliomas" recorded in the literature where a revision of such diagnosis has been made or seems indicated by the later course of the case; however, the possibility of late or distant metastasis to adjoining nodes or viscera or the development of similar nonchromaffin paragangliomatous tumors in other organs of the parasympathetic glomus system must be recognized.

SUMMARY.

1. Three cases of glomus jugulare tumors are herein reported together with photomicrographs of the lesion.

2. A review of the pathogenesis, salient clinical features and management is given.

3. It would seem that a critical review of all middle ear and mastoid tumors with a diagnosis of angioma, angiofibroma or endothelioma, perithelioma, adenoma, xanthomatous polyp, or arteriovenus aneurysm should be undertaken, as was done in our cases, with the idea of possible revision of such diagnosis to that of glomus jugulare tumor where the histologic picture so justifies.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSES.

The 1952-1953 Home Study Courses in the basic sciences related to opththalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1, 1952, and continue for a period of 10 months. Detailed information and application forms can be secured from Dr. William L. Benedict, executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn. Registrations should be completed before Aug. 15.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

March 1, 1952.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F; Model H; Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chcago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2.
Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.
Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2.
Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).
Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.
Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Rochester Model R-1; Rochester Model R-2.
Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model 103BM.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model J-92.
Manufacturer: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.
Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.
Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Vice-President: Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Meeting: Royal York Hotel, Toronto, Canada, May 18-19, 1952.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

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Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Royal York Hotel, Toronto, Canada, May 23-24, 1952.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President Dr. C. Stewart Nash, 108 Medical Arts Bldg., Rochester, N. Y.
Meeting: Royal York Hotel, Toronto, Canada (mornings only), May 20-22, 1952.

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Palmer House, Chicago, Ill., October 6-10, 1952.

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AMERICAN COLLEGE OF SURGEONS
SCHEDULE OF SECTIONAL MEETINGS—1952

The Vancouver Hotel, Vancouver, B. C., Monday, Tuesday, Wednesday, March 31, April 1, 2.

The Pioneer Hotel, Tucson, Ariz., Monday, Tuesday, Wednesday, April 7, 8, 9.

Royal York Hotel, Toronto, Canada, Thursday, Friday, Saturday, May 15, 16, 17.

Those who plan to attend a Sectional Meeting may select the meeting which in time or place is most convenient.

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Meeting: Palmer House, Chicago, Ill., Oct. 11, 1952.

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Meeting: Palmer House, Chicago, Ill., Oct. 17, 1952.

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Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: January, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

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Meeting: St. Louis, Hotel Jefferson, Oct. 1-3, 1952.

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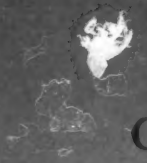

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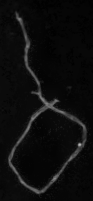
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